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FOUNDED BY JAMES PLEASANT PARKER

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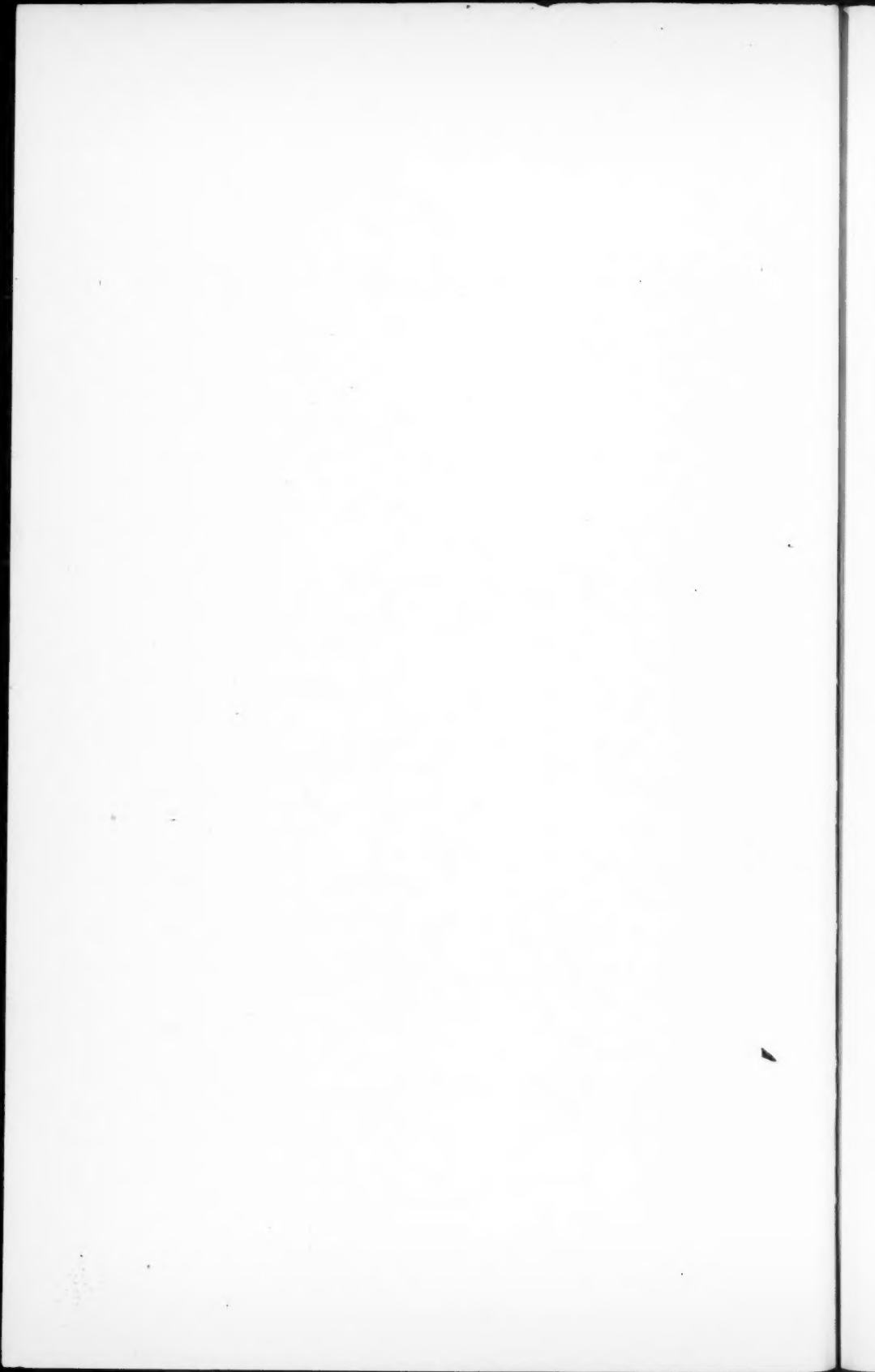


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INCORPORATING THE INDEX OF OTOLARYNGOLOGY.

VOL. XLII.

MARCH, 1933.

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I.

CICATRICAL STENOSIS OF THE LARYNX.*

LOUIS H. CLERF, M. D.,

PHILADELPHIA.

Stenosis of the larynx may occur in the course of certain infections or may result from various forms of trauma. The chief cause, however, is improperly performed tracheotomy. In practically all of the cases of acute laryngeal stenosis requiring tracheotomy for relief of dyspnea no difficulty is encountered with decannulation after the acute process has subsided, provided a proper tracheotomy was done and adequate after care was instituted. Failure to decannulate is most often observed in cases of high tracheotomy or faulty after care. Among the cases of chronic stenosis of the larynx sent to Jackson for treatment it was found that in approximately one-sixth of these there had been necrosis of the cartilaginous framework of the larynx, probably caused by the primary condition for which tracheotomy was done, while in the remaining five-sixths the stenosis was due either to faulty tracheotomy or to improper after care. Observations of other writers have corroborated these findings.

In a series of twenty-eight cases of cicatricial stenosis of the larynx sent to the Bronchoscopic Clinic and treated by some form

*Read before the Meeting of the Academy of Ophthalmology and Otolaryngology, September 20, 1932, Montreal, Canada.

of external surgical procedure, it was found that tracheotomy had been performed either through the cricoid cartilage or at a higher level in twenty-one instances. None of these cases was suitable for the more conservative methods of treatment after a low tracheotomy had been performed. In sixteen cases the airway was completely occluded by a cicatricial barrier.

The following tabulation indicates the primary conditions producing dyspnea for which tracheotomy was done and the frequency of high tracheotomy and atresia of the larynx in each group:

Primary Condition	Number Cases	High Tracheotomy	Atresia Larynx
Diphtheria	13	8	12
Typhoid fever	1	1	1
Influenza	1	1	0
Trauma	8	6	1
Papilloma (radium)	3	3	2
Bilateral laryngeal paralysis (post- thyroidectomy)	2	2	0
Total.....	28	21	16

Diphtheria.—The influence of intubation in laryngeal diphtheria on the incidence of cicatricial stenosis is debatable. Sewall states that in the acute inflammatory conditions, associated with diphtheria, laryngeal stenosis occurs where tracheotomy has been done without intubation almost as frequently as where the patient has been intubated. Properly performed intubation or tracheotomy very probably are not often followed by stenosis. Faulty tracheotomy unquestionably is the most common cause of chronic stenosis. Of the thirteen cases reported as following diphtheria, twelve had atresia. Nine cases had been intubated during the disease and later tracheotomy was done; in two, high tracheotomy had been done without preliminary intubation, and in the remaining case the tube had been properly placed below the second tracheal ring. In the nonatresic case a high tracheotomy had been performed. It is interesting to note the effects of aspiration of secretion and membrane, together with the prophylactic measures employed in diphtheria prevention, on the incidence of intubation in laryngeal diphtheria. At the Philadelphia Hospital for Con-

tagious Diseases,* direct laryngoscopic aspiration has been practiced since 1924. The percentage of intubation cases in 1923 was 26.74. Although there was an increase in the number of intubated cases in 1924, Dr. Lucchesi stated that during the period from 1925 to 1928 inclusive, the rate dropped to 15.5 per cent (average for four years). Beginning in 1929, there was a definite increase in the number of cases intubated. He attributed this to the admission of a large number of cases of acute laryngitis caused by the streptococcus, staphylococcus and other organisms as cases of laryngeal diphtheria. The policy employed at the present time consists of direct laryngoscopic inspection of the larynx with aspiration only of those cases presenting clinical evidences of diphtheria. Neither aspiration nor intubation is practiced in the cases of nondiphtheritic laryngitis.

Papilloma.—The employment of conservative methods of treatment in papilloma of the larynx results in a normal larynx when the growths cease to recur. Direct application of radium into the larynx, as was practiced in the three cases reported, is not only useless in papilloma but is commonly followed by extensive perichondritis and necrosis of the laryngeal cartilages (Clerf). In two of the cases there was an atresia of the airway with destruction of the cricoid cartilage. In one case the papilloma recurred in spite of the fact that little of the normal larynx remained.

Laryngeal Paralysis.—Abductor paralysis of the vocal cords following thyroidectomy is not productive of cicatricial stenosis. Emergency tracheotomy is often resorted to, however, and when hastily performed is often improperly done. With the thyroid gland removed the trachea is more accessible in these cases and tracheotomy should be more easily performed.

Trauma.—Extensive injury often with a compound fracture of the larynx is a relatively common cause of cicatricial stenosis and is apparently becoming more frequent with the increasing number of automobile accidents and other injuries. Urgent dyspnea requiring prompt tracheotomy, often improperly performed, and the occurrence of perichondritis and necrosis of cartilage in compound fractures communicating with the airway, are the most

*Personal communication from Dr. P. F. Lucchesi, Acting Superintendent, Philadelphia Hospital for Contagious Diseases.

common causes of chronic stenosis in this group. (Fig. 1.) In four of the eight cases reported the tracheotomy tube was inserted through the cricothyroid membrane, and in two the tube was introduced through an opening made in the larynx at the time of the injury. (Fig. 2.) Very satisfactory results can be secured and serious stenosis avoided in cases of fracture if a low tracheotomy is performed early and proper surgical measures are employed in the treatment of the injured area (Mullen).

Diagnosis.—A diagnosis of cicatricial stenosis of the larynx can usually be made without difficulty. In adults, mirror laryngoscopy may be sufficient. In children, direct laryngoscopy is necessary. Certain cases may require bronchoscopy or retrograde laryngoscopy, through the tracheotomy opening (Tucker). Occasionally it may be difficult to determine if an airway is present. Carefully performed direct laryngoscopic examination, palpating the cicatricial barrier with a filiform bougie, will often prove helpful. If the tracheotomy has been performed through the thyroid cartilage or the cricothyroid membrane the tube should be momentarily removed during the examination, for in this high position it may completely occlude the airway. Roentgen studies should always be made to rule out foreign body and also for such additional information as may be secured by this method of examination.

Treatment.—Before proceeding with a plan of treatment all possible sources of infection about the upper air and food passages should be investigated and removed (Orton). General systemic studies should be carried out and syphilis should be excluded. The lungs should be carefully investigated. The presence of a pulmonary infection, as bronchiectasis, would contraindicate any form of surgical treatment. The form of treatment to be instituted depends on the degree of stenosis and the condition of the laryngeal cartilages. These data are secured at the time of the diagnostic study and are obtained by laryngoscopy, bronchoscopy, esophagoscopy, Roentgen studies and palpation of the neck.

In minor degrees of stenosis, with little or no involvement of the supporting laryngeal framework, one may consider some form of dilatation. Low tracheotomy should always be performed. Many methods of dilatation, including metal plugs and rubber

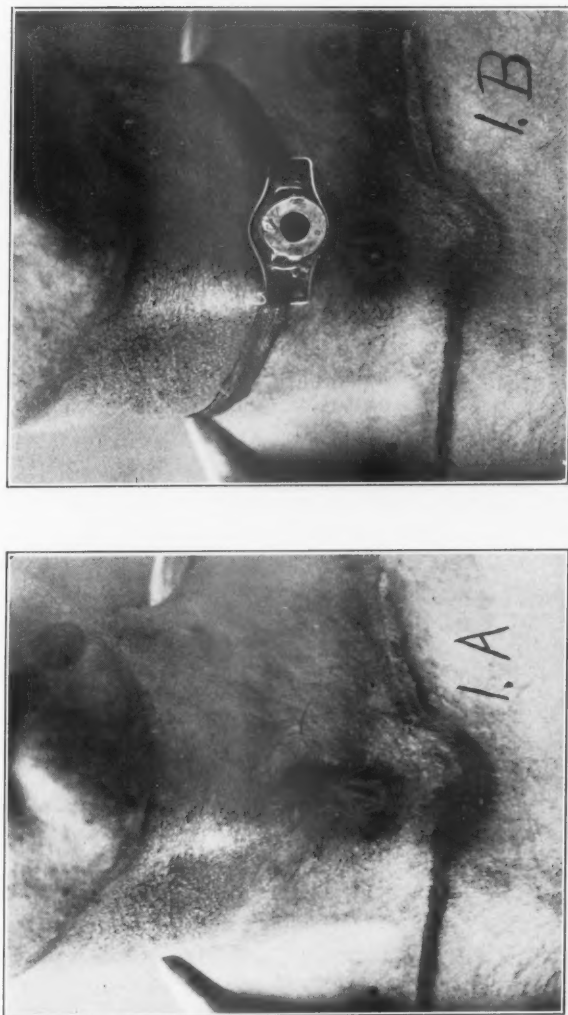


Fig. 1. Case of a patient with atresia of the airway following a compound fracture of the larynx. The tracheotomy tube, in a high position, as shown in its relation to the line indicating the upper border of the clavicles and suprasternal notch had been inserted into the airway through an opening made by a stick of wood striking the neck (A). The tracheotomy tube, an unorthodox type, had been changed but once in fourteen months and exhibited considerable crusting of secretions (B).



Fig. 2. An opening accidentally made through the crico-thyroid membrane and lower border of the thyroid cartilage during a stabbing affair served as a fistula for an improvised rubber tube airway. A tracheotomy tube had been worn for about two weeks but was removed to be replaced in several days by the rubber tube held in situ by a safety pin and cotton string. After four months there was found a marked cicatricial stenosis of the larynx which was successfully treated by the translaryngeal fixation method.

tubes (Von Schroetter), intubation tubes (Leffert) and various modifications have been recommended. In my experience direct laryngoscopic dilatation, using metal bougies as recommended by Jackson, has proven satisfactory. Good results have been secured by some observers with progressive dilatation, using metal dilators through the tracheotomy opening (Thost-Bruggemann). This method is of value only in selected cases.

In cases of atresia and in those cases of nonatresic stenosis that do not respond to direct laryngoscopic methods of dilatation more radical treatment is necessary. With the development of satisfactory methods of surgical treatment I do not favor the use of intubation tubes or similar apparatus that is introduced into the larynx through its natural opening and retained in position by mechanical means. Cases responding to these methods would usually respond to direct laryngoscopic dilatation.

The underlying principles of surgical treatment are restoration of the airway by removal of obstructing tissue and its maintenance by mechanical means during the process of cicatrization and epithelialization.

One of the great obstacles encountered in the treatment of these cases lies in the fact that often there is an absence of the supporting cartilaginous framework so necessary in maintaining an open airway. Involvement of the cartilage by the primary disease or secondary infection following high tracheotomy is commonly followed by loss of normal cartilage and its replacement by firm fibrous tissue or an attempt at the formation of new cartilage. Proliferation of cartilage is most commonly seen in those cases where the posterior lamina of the cricoid is involved. This newly formed tissue consists almost entirely of a heaping up, on the posterior wall, of fibrous tissue elements containing irregularly distributed islands of cartilage with occasional areas of new bone formation. (Fig. 3.) This tissue cannot be utilized in the reconstruction of the larynx but must be removed in order to secure an adequate airway. Its continued proliferation often interferes with retention of the dilating apparatus at this point. Regeneration of a normal cartilage in these cases has never been observed.

Until recently the commonly advocated method of treatment consisted of packing the newly established airway for a time, followed by the introduction of some form of dilating apparatus and a Jackson laryngostomy tube. Various modifications of tubes (Tucker) and other apparatus have been used. In extensive removal of cicatricial tissue the use of skin grafting has aided in hastening epithelialization (Arbuckle).

While excellent results were obtained in many cases by laryngostomy, particularly when compared with the difficulties en-

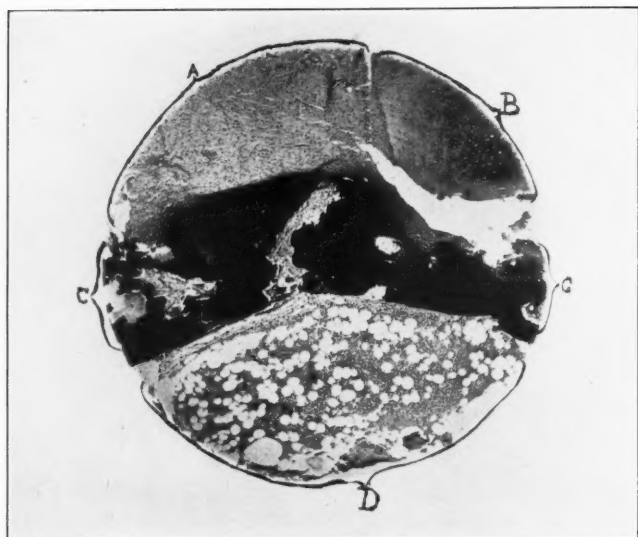


Fig. 3. 46X. Photomicrograph made of tissue removed from the larynx in the case of a child, aged 10 years. In this case the anterior portion of the cricoid and a part of the thyroid cartilages had been destroyed and were replaced by fibrous tissue. The posterior lamella of the cricoid was replaced by a mass of newly formed connective tissue as shown above. This completely occluded the airway necessitating its removal before proceeding with the translaryngeal fixation of a rubber tube. (A) Dense fibrous tissue which lacks blood vessels and is devoid of any inflammatory reaction; (B) Area of typical cellular hyperplastic hyaline cartilage; (C) Typical bone formation; (D) Cellular marrow tissue showing normal characteristics. (Slide by Dr. B. L. Crawford.)

countered in the earlier methods of treatment, this method nevertheless left much to be desired. Frequent changing of apparatus, which was often painful, time consuming and disagreeable; the problems involved in treating young children who fail to co-operate and the danger of injury to cartilage, the necessity for wearing the apparatus for months and even years, all added to the problems involved in the treatment of this condition. In addition there was always a question regarding the outcome in cases with extensive cartilage destruction.

These problems induced Schmiegelow to devise a new method of treatment of cicatricial stenosis of the larynx. Since 1910 he had successfully employed this method and had treated eighteen cases with satisfactory results. The method consists of laryngofissure, removal of webs, strictures and obstructing scar tissue, restoring the laryngeal lumen to as nearly normal as possible and the translaryngeal fixation of a rubber tube introduced through the fissure in the larynx. (Fig. 4.) He found that the tube caused little inconvenience to the patient; there was no reaction of consequence; changing of the apparatus, a serious problem in children, was unnecessary and the duration of treatment and hospitalization was shortened. In adults, where one can introduce a large rubber tube in the larynx, he dispensed with the tracheotomy tube. In children, however, he recommended that the tracheotomy tube be left in, because of the danger of obstruction of the rubber tube by food particles. Age was no contraindication to the use of this method. Three of his patients were less than three years of age. The period of treatment in some of his cases was less than one month.

After using the Jackson method of laryngostomy in fifteen of the twenty-eight cases reported and noting some of the objections previously mentioned, I decided to employ the translaryngeal fixation method devised by Schmiegelow. Of the thirteen cases treated by his method six were under 10 years of age, the youngest being $2\frac{1}{2}$ years. The method employed is essentially that advocated by Schmiegelow. In the earlier cases overflow of secretion into the trachea proved troublesome and necessitated closure of the upper end of the transfixing tube by direct laryngoscopic introduction of a rubber cork. I now routinely close the upper end of the tube and all patients wear a tracheotomy cannula. They do not object to this since all wore a tube before operation and in many the larynx was atresic. It is of interest to note that a rubber tube may be left in situ for several months without a marked reaction of the laryngeal tissues or incrustation about the tube. In one case the tube was allowed to remain fifteen weeks without the development of unfavorable signs.

Several points in the operation are worthy of comment. The laryngeal or tracheal airway should be made as nearly normal



Fig. 4. A case of cicatricial stenosis at the cricoid level following a compound fracture of the larynx. In Fig. A is seen the shadow of the rubber tube transfixed with a silver wire. In the lateral view (B) the relation of the end of the tube to the hyoid bone can be seen. This was one of the early cases and the upper end of the rubber tube was left open. The placement of the silver wire was not symmetrical and was also at a low level.

as possible by removal of all obstructing tissue. The rubber tube, preferably of pure gum rubber, should be sufficiently large to fit snugly in the larynx. It should extend above and below the limits of the stenosis. If allowed to remain open at its upper end it should not extend above the upper orifice of the larynx. A gauge 19 silver wire on a large curved cutting edge needle is used to transfix the tube, passing through the wings of the thyroid cartilage. The ends of the wire should be cut sufficiently short so that they may retract beneath the skin surface. Slight infection of the wound or at the point of entrance of the silver wire through the skin may occur but is not troublesome. Hospitalization in the case of adult patients need not be continued more than several weeks since the wound heals promptly.

No definite rules can be formulated with regard to the duration of time that the rubber tube should remain in situ. In cases with no cartilage loss the tube may be removed in four to six weeks. I have removed none under six weeks and, in cases with considerable cartilage loss, have allowed it to remain for three to three and one-half months. Removal of the tube is accomplished by direct laryngoscopy, using a simple form of grasping forceps. The silver wire offers no resistance to removal.

In long standing cases of stenosis with small, poorly developed larynges it may be necessary to do a second operation to introduce a larger rubber tube into the larynx. In one case that had been unsuccessfully operated on elsewhere by the old method of laryngostomy, an 8 mm. tube only could be transfixed in the larynx. At a subsequent operation a 12 mm. tube was substituted for this.

In cases of extensive loss of cartilage there may be some difficulty in retaining a soft rubber tube in the larynx. I have found it satisfactory to support the rubber tube with a solid rubber plug, as shown in Fig. 5.

SUMMARY OF CASES.

A series of twenty-eight cases of cicatricial stenosis of the larynx found unsuitable for direct laryngoscopic dilatation is reported. All of the patients had been tracheotomized for relief of dyspnea. In twenty-one, high tracheotomy had been per-

formed; in sixteen there was an atresia of the airway. Of the thirteen cases of postdiphtheritic stenosis, twelve were atresic.

Fifteen cases were treated by laryngostomy, followed by packing the larynx and using various forms of tube dilators. Seven of these cases were atresic and in nine a portion of the cricoid cartilage had been destroyed. One case became discouraged and

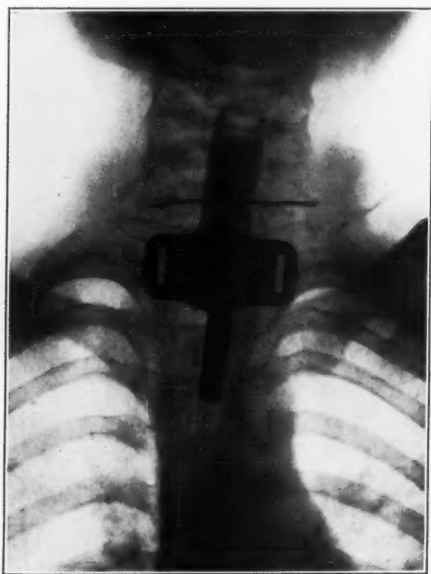


Fig. 5. Case of postdiphtheritic stenosis of the larynx in a girl aged 12 years. Because of the marked loss of cartilaginous support, the rubber tube was supported by a solid rubber plug the shadow of which is seen to terminate about one centimeter from the upper end. In this case the silver wire has been properly placed.

discontinued treatment. Three have not been cured, although an epithelialized airway is present. There is lacking sufficient cartilaginous support to maintain an adequate airway. These cases will probably be treated by the translaryngeal fixation method in combination with supporting transplants, although one has already received bone transplants. Two cases have been improved and

decannulated but remain dyspneic on exertion. Nine cases can be considered as cured; they have been decannulated and have ample airways and satisfactory voices.

Thirteen cases were treated by the Schmiegelow method. Nine of these had atresia of the larynx; in ten there was absence of a portion of the cricoid; in three of these there was also some injury to the thyroid cartilage. Nine of these cases have been cured; one will require a second operation for the introduction of a larger tube; three are still under treatment and cannot be classified so far as end results are concerned.

CONCLUSIONS.

Improperly performed tracheotomy and inadequate after care are the most common factors in the etiology of cicatricial stenosis of the larynx. Recognition of this fact is of great importance in prophylaxis.

The translaryngeal fixation operation seems to be the method of choice in the treatment of cases of cicatricial stenosis of the larynx that cannot be treated by the more conservative methods. This procedure obviates the need for frequent, time consuming, painful dressings and changes of apparatus. The duration of treatment and the period of hospitalization are shortened. Results, even in cases where there is a loss of the normal supporting tissues of the larynx, are uniformly good.

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II.

THE HISTOLOGY AND CHRONIC INFLAMMATION OF THE NASAL MUCOUS MEMBRANE.*

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Histology.—The essential tissues comprising a mucous membrane are (1) a surface epithelium, (2) a basement membrane and (3) a tunica propria or stroma. In addition, there may be (4) a muscle coat, the muscularis mucosa and (5) a submucosa. Of the mucous membranes of special interest to the rhinolaryngologist only the esophagus and lacrimal duct possess these additional coats.

The epithelium of the mucous membrane may be of any of the varieties of epithelial cells. According to the shape of the cell it may be classified as squamous, cuboidal, columnar or transitional. According to the arrangement of the cell it may be stratified or pseudostratified.

The basement membrane is a band of unstained or faintly staining tissue, collagenous in character, on which the epithelial cells apparently rest. Its origin is uncertain. From the fact that it takes the connective tissue stain it is thought to be derived from the underlying connective tissue. It varies in thickness; it may be so delicate as to appear absent; in chronic infections it may be tremendously thickened. It is cribriform. These canaliculi permit processes of the underlying connective tissue cells to extend into the epithelium and they permit passage of cells to the surface.

The tunica propria or stroma consists of a fibro-elastic connective tissue network, which serves to attach the membrane to the underlying structures. Superficially the stroma is loose, rich

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I wish to express my appreciation to Dr. Werner Mueller for the microphotographs illustrating this paper and to Dr. P. E. Ireland for the colored slides.



Fig. 1. Nasal fossæ of 4 months fœtus. The epithelium is of the pseudo-stratified ciliated variety.

in cells, but as it deepens it becomes more compact and condensed, with the progressive appearance of elastic fibers. Over bone or cartilage this condensation of the stroma forms, respectively, the periosteum or perichondrium.

Of the cellular elements, the lymphocyte predominates. These may be scattered throughout the tissue as diffuse lymphoid tissue, or may be gathered into a compact mass to form a distinct lymph node or nodule.

The stroma supports the glands. These vary, from the simple straight tubule lined with goblet cells, to the tubo-alveolar type of gland.

The blood supply is by way of vessels which enter deep in the stroma. These vessels send branches through the propria to form a dense capillary network beneath the epithelium and around the



Fig. 2. Nasal fossæ of an 8 month child. The epithelium is of the ciliated variety.

neighboring glands. The venous return is by way of superficial blood spaces which lead to a deeper venous plexus. In certain areas of the respiratory mucosa these plexuses are so greatly developed as to form cavernous sinuses and assume the character of erectile tissue.

Such then is the basic histologic picture of any mucous membrane. The nasal mucosa shows characteristic variations according to site.

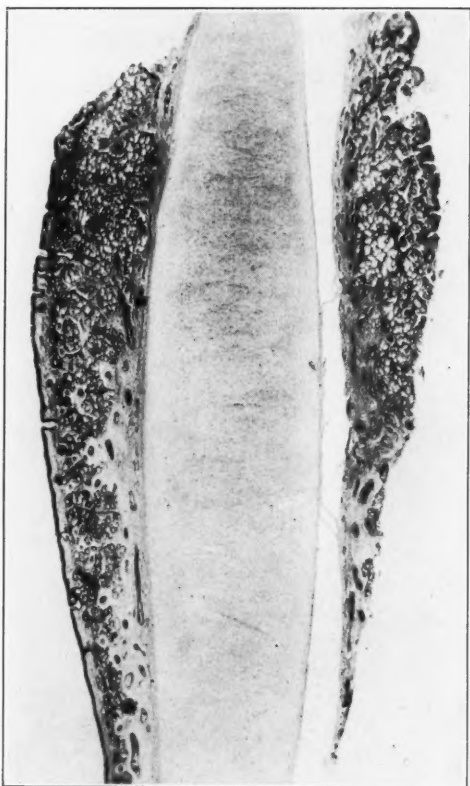


Fig. 3. Nasal septum—adult. Characterized by many glands in the stroma, particularly in the region of the tubercle.

The Nasal Septum.—In the embryo and infant, the epithelium is of the pseudostratified ciliated variety. In the adult, due to the traumatization incident to life, the exposed portions of the septum, frequently shows nature's protective measure, in that the pseudostratified ciliated epithelium is replaced by the stratified squamous variety. The basement membrane is distinct. The stroma is characterized by the tremendous number of mucous and serous glands. In the region of the tubercle of the septum

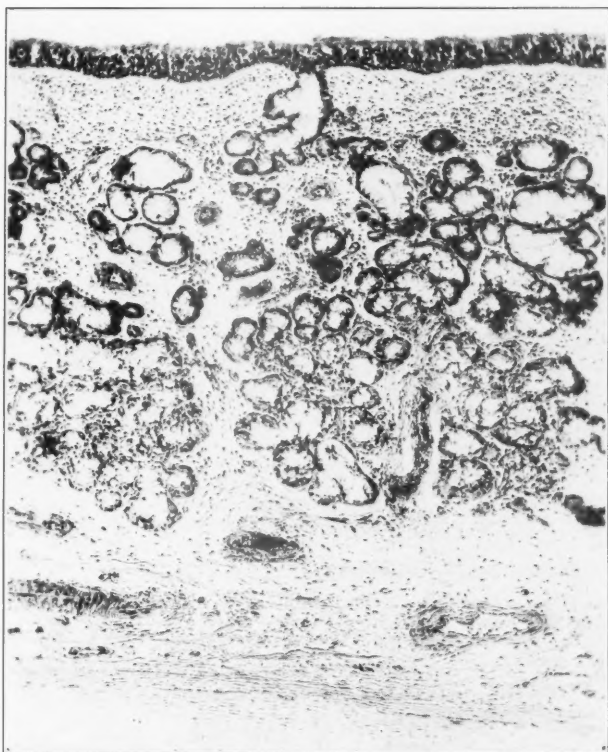


Fig. 4. Nasal septum, 75 \times , region of tubercle, showing the profusion of g'l'ands.

there appear large blood lakes or cavernous spaces. Lymphocytic infiltration of the stroma is marked. In places the lymphoid cells form distinct lymph nodules.

Over the cartilage and bone, the stroma becomes condensed to form the perichondrium or periosteum. At the junction of the cartilage and bone, fibers of the stroma interlace with fibers from the opposite side.

The epithelium of the olfactory portion is that of the stratified variety, the surface cells being both sustentacular or supporting cell and the olfactory cell.

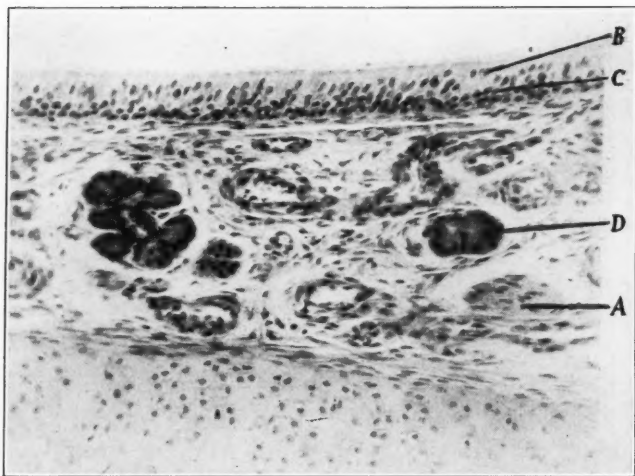


Fig. 5. Olfactory mucous membrane of cat. A. Nerve Bundle. B. Oval nuclei. C. Round nuclei. D. Glands of Bowman.

The sustentacular cell presents three distinct parts:

(1) A superficial portion, which is broad and cylindrical, containing pigment and granules arranged in longitudinal rows. These cells have well marked straight thickened free borders, which unite to form the so-called "membrana limitans olfactoria."

(2) A middle portion which contains an oval nucleus. As the nuclei of these cells all lie on the same plane, they form a distinct narrow band—"the zone of oval nuclei."

(3) A thin filamentous process which extends from the nuclear portion down between the cells of the deeper layers.

The olfactory cells lie between the supporting cells. Their nucleus is spherical, they lie at different levels and are more deeply placed than those of the sustentacular cells; they form the so-called "zone of round nuclei." The olfactory cell sends a dendrite which extends to the surface, and an axone, which collects with other axones to form eighteen to twenty-four bundles to pass through the cribriform plate to the olfactory bulb.

Extending from the surface cells to the basement membrane are the basal cells. They are small, nucleated cells, with irregular

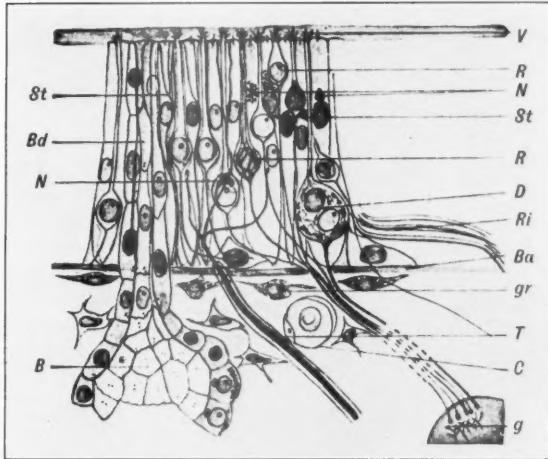


Fig. 6. Diagram of the olfactory mucous membrane of man: *St*, Sustentacular cells with tonofibrils; *R*, olfactory cells with neurofibrils and the olfactory vesicles; *V*; *D*, large binucleated olfactory cell; *Ri*, olfactory fibers; *T*, myelinated fiber of the trigemius; *g*, olfactory glomerulus; *B*, gland of Bowman, with diploemes in its cells, and with its excretory duct; *Bd*; *gr*, granular connective tissue cells; *Ba*, layer of basal cells; *N*, Golgi net in the sustentacular and in the olfactory cells; *C*, capillary. After Kolmer.

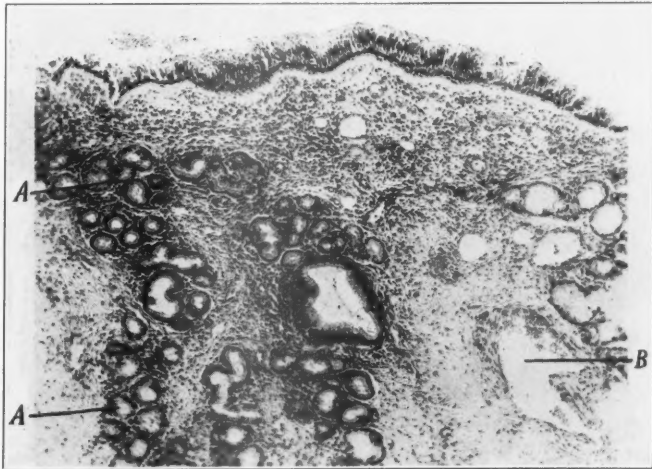


Fig. 7. Middle Turbinate, 100 \times . *A*. Glands. *B*. Cavernous sinus.



Fig. 8. Inferior turbinate with dilated smell bodies. 30 \times .

or branching protoplasm which anastomoses with that of neighboring basal and sustentacular cells.

The basement membrane of the olfactory portion is not well developed.

The tunica propria is delicate, loosely arranged. Embedded in the stroma are a large number of simple branched glands—the "glands of Bowman." The secretory cells of these glands are large and irregular and contain a yellowish pigment. These glands, described as serous, are now believed to be mucous.

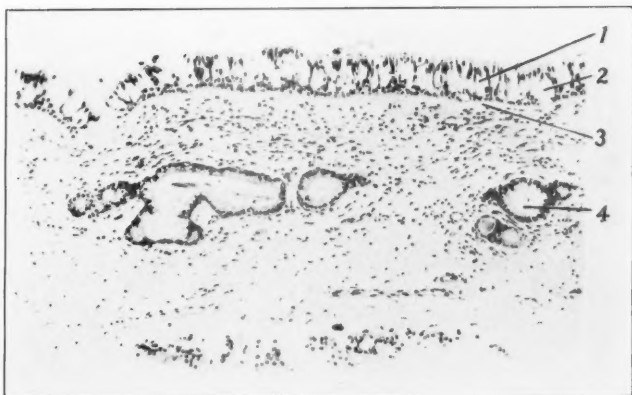


Fig. 9. Normal maxillary sinus mucosa. 100 \times . 1. Pseudostratified, ciliated epithelium. 2. Goblet cells. 3. Basement membrane. 4. Glands.

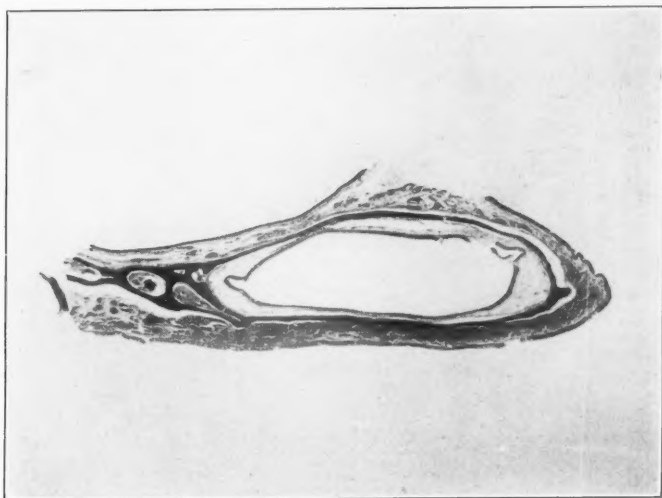


Fig. 10. Ethmoid cell, 10 \times .

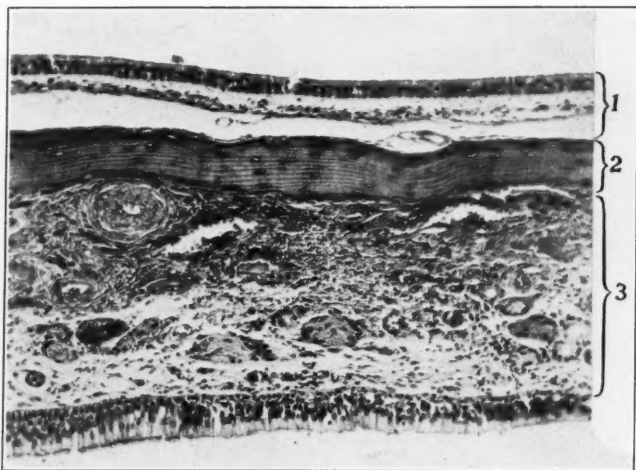


Fig. 11. Ethmoid, 140 \times . 1. Mucosa of ethmoid. 2. Bone.
3. Nasal mucosa.

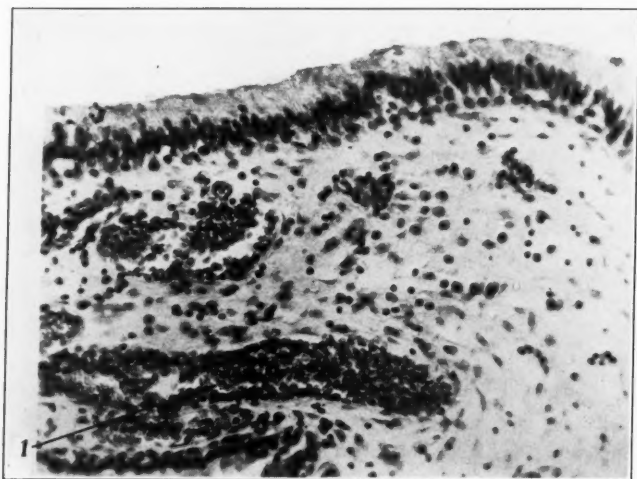


Fig. 12. Antral mucosa, 300 \times . Acute inflammation.
1. Dilated capillary.

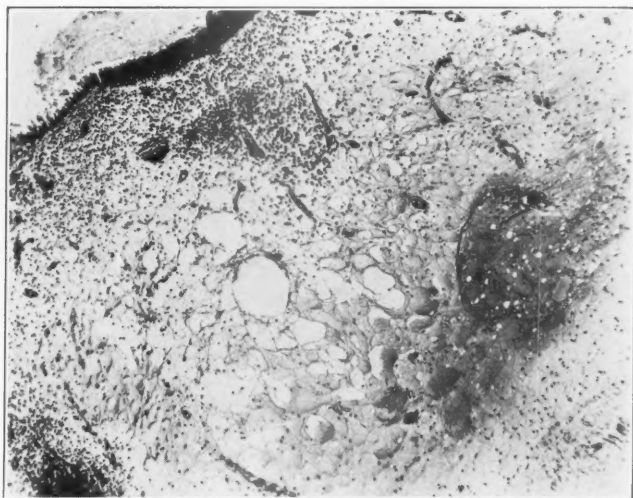


Fig. 13. Edematous mucous membrane.

The Middle Turbinate.—The mucosa of the middle turbinate varies greatly in thickness. The ciliated epithelium is frequently replaced by a low cuboidal or squamous variety over the anterior tip. The stroma is particularly rich in glands, especially over the posterior tip of the turbinate. The periosteum is firmly attached to the underlying bone.

Inferior Turbinate.—Like the middle turbinate, the epithelium of the anterior tip and inferior border of the inferior turbinate in the adult, may be replaced by a low cuboidal or squamous epithelium. The basement membrane is usually distinct. The stroma is characterized by the pronounced number of blood lakes or cavernous sinuses. Glands are likewise numerous, but not to the extent of the middle turbinate.

Maxillary Antrum.—The mucosa of the normal maxillary sinus is a thin, delicate structure. The epithelium is continuous with that of the nose—of a pseudostratified ciliated variety, containing numerous goblet cells. The basement membrane is fairly distinct. The stroma is poorly cellular. The glands are scattered, but are

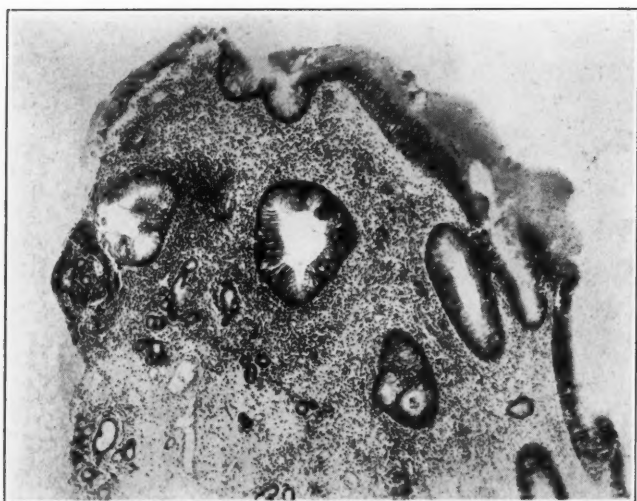


Fig. 14. Chronic sinusitis, 60 \times . Infiltrative type, showing marked cellular reaction.

most numerous in the region of the osteum. The periosteum strips readily from the underlying bone.

Ethmoid.—The mucous membrane of the ethmoid cells resembles that of the maxillary sinus. The epithelium is likewise ciliated columnar variety with an occasional goblet cell. The stroma is thin and delicate, containing but a few glands. It is more firmly attached to the underlying bone than that of the maxillary antrum.

Frontal, Sphenoid.—The mucosa of the frontal and sphenoid is very similar to that of the ethmoid. Stratified, ciliated epithelium, faint basement membrane, stroma delicate, few glands but fairly firmly attached to the underlying bone.

Pathology.—Such then is the histologic picture of a normal nasal mucous membrane. Let us review the changes that occur when this normal is altered by inflammation. First, let us limit and simplify our definition of inflammation as "the tissue reaction to an irritant." The changes that result are (1) those of the blood stream and (2) those of the cellular elements.

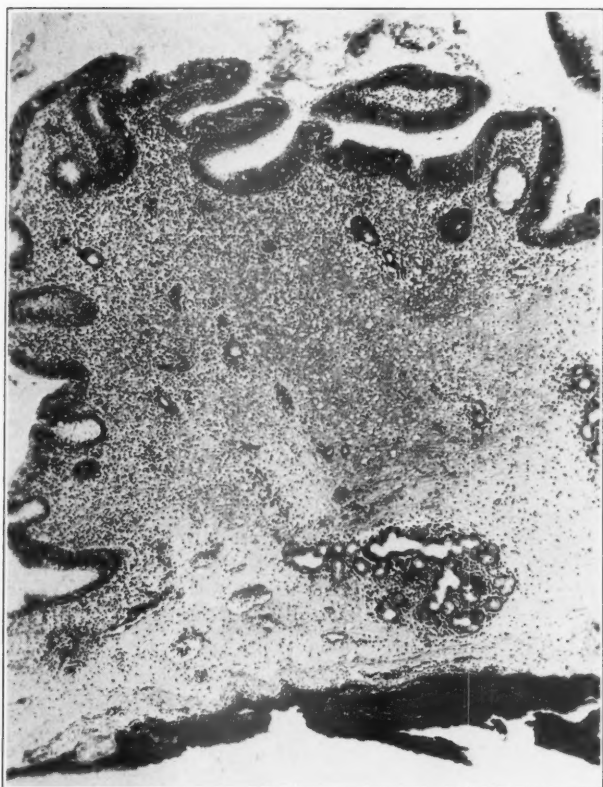


Fig. 15. Chronic sinusitis, 45 \times . Polypoid infiltrative type.

Most pathologists are agreed that the alterations in the blood stream are first a temporary constriction of the vessel with an acceleration of the blood stream. This is followed by a dilatation of the vessel and subsequent slowing of the current of the blood.

Examination of the blood as it flows along through the vessels shows that there is normal central, axial zone, containing the blood corpuscles; and, in addition, there is, between the axial zone and the capillary wall, a peripheral zone containing blood

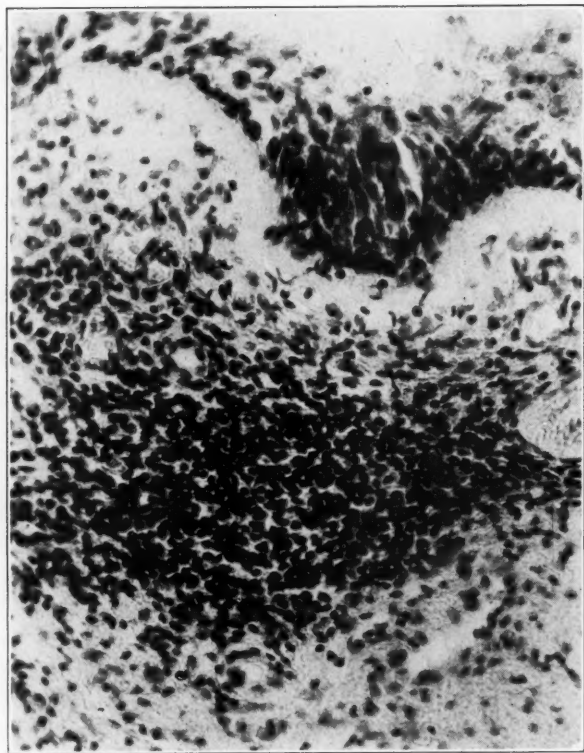


Fig. 16. Chronic infiltrative sinusitis, 300 \times . Predominating cell being the lymphocyte with an increase of plasma and connective tissue cells.

plasma. These zones are maintained by the vertical force acting upon the heavy cells as compared with the lighter plasma. In normal circulation or in the early stages of inflammation, when the vessel wall is constricted, these zones are clearly maintained. With the dilatation of the vessel wall and slowing of the circulation, the lighter cells of the blood stream, due to the lowered specific gravity, appear in the peripheral zone. During this stage the leucocytes can be seen lagging along the vessel wall adhering,

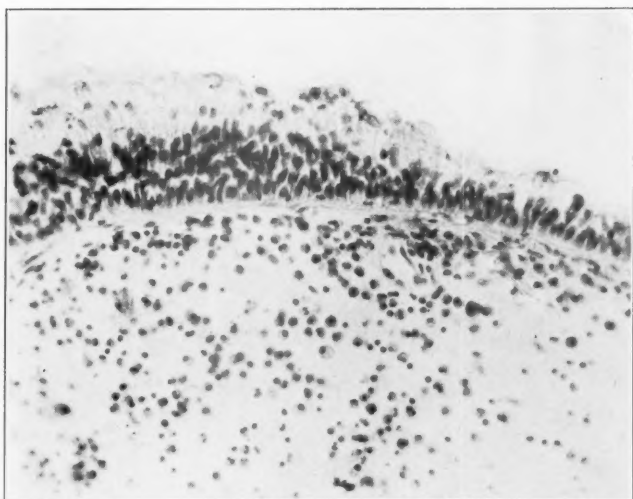


Fig. 17. Chronic sinusitis, asthmatic, 300X. Great increase of the eosinophiles.

and finally by diapedesis pass through the wall into the surrounding tissue. There they migrate toward the point of injury. At the same time the fluid of the blood stream passes through the vessel wall and appears in the tissues. This then is the first reaction of inflammation. The fixed tissues react to the inflammation by a migration of the fixed tissue cells, particularly the connective tissue cells.

In mucous membranes, as there is a dense capillary network just beneath the basement membrane, it appears reasonable to expect that the most marked change occurs primarily in this region. In acute inflammations of mucous membrane there is, therefore, a dilatation of the tissue or lymph spaces just beneath the epithelium, with an increase of the cellular elements. As the infection passes from the acute to the chronic stage the polynuclears disappear, so that of the cellular elements the small lymphocyte predominates.

The tissue reaction of chronic inflammation is, therefore, a thickening of the blood vessel wall, with an increase of the cellu-

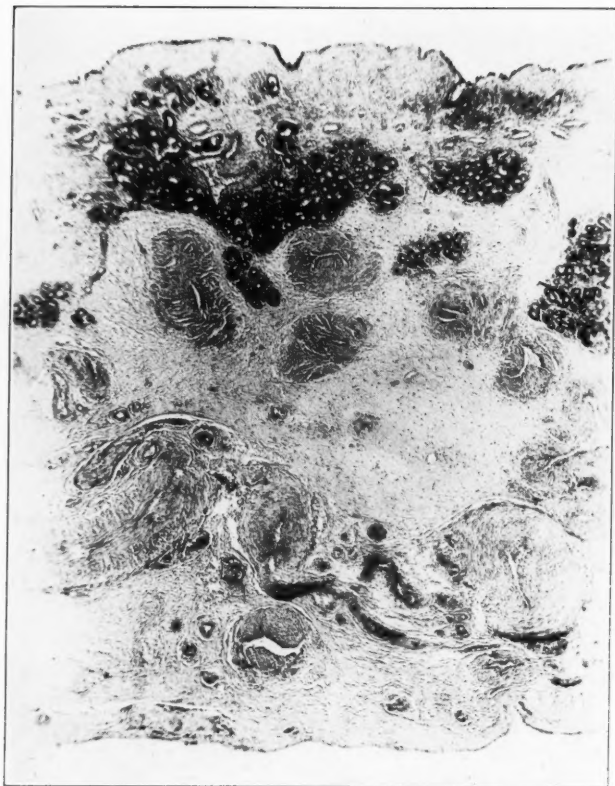


Fig. 18. Chronic sinusitis, 300 \times . Fibrosis.

lar elements of the tissue. These cellular elements are the lymphocytes, plasma cells, connective tissue cells and endothelial cells. In some conditions, such as asthma, hay fever and the parasitic infections, the eosinophile may show a marked increase in number.

I agree most heartily with Mullin and Ball¹ in that the pathologic findings of chronic sinusitis give no clue as to the clinical history of the case. According to the microscopic picture, I have classified chronic inflammation of the sinus mucous membrane—

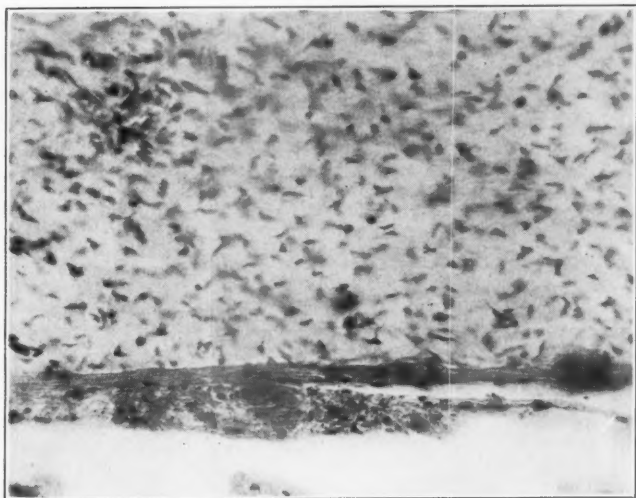


Fig. 19. Chronic sinusitis, 300 \times . Fibrosis.

1. Edematous.
2. Infiltrative.
3. Fibrotic.
4. Cystic.
5. Degenerative.

Edematous.—The predominant microscopic picture is, as the name indicates, that of edema. This edema is most marked, as would be expected from the dense capillary network beneath the epithelium, in the superficial portion of the stroma. The cellular elements are not prominent. The vessel walls are thickened and the glands are dilated.

Infiltrative.—In the infiltrative type of chronic inflammation, there is a cellular predominance consisting principally of the lymphocytes. This infiltration is throughout the stroma, particularly beneath the basement membrane, but is most marked around the glands. In certain areas the lymphocytic infiltration is so pronounced as to form or resemble lymph nodules. Most textbooks speak of this lymphocytic infiltration as being most marked

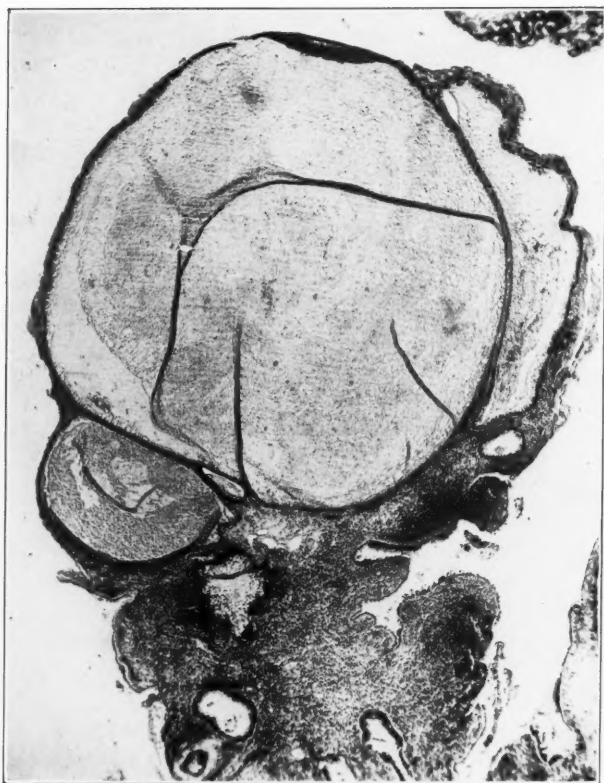


Fig. 20. Chronic sinusitis, 25 \times . Cystic.

around the blood vessels. It is true that there is a perivascular infiltration, but I have been very much impressed by the marked periglandular infiltration.

In the chronic inflammations the glands are exceedingly numerous. Whether this is an actual increase in glands or a great activity of glands which are normally present so that they are dilated and easily recognized, I am unprepared to say. The blood vessels show thickening.



Fig. 21. Chronic sinusitis, 250 \times . Showing infected cyst.

Fibrotic.—In the fibrotic type of chronic inflammation the fibrosis predominates. There is a marked increase of fibrous tissue with constriction of the glands and a marked decrease of the cellular elements. I have been unable to find many specimens showing minute abscesses deep in the stroma, as reported by some authors.

Cystic.—The subject of cysts of the antral mucous membrane has been covered most thoroughly by McGregor.² Whether these



Fig. 22. Fibrotic turbinate with cyst formation.

cystic changes are due to a blocking of the ducts of the glands by occlusion of the lumen or by a choking of the lumen by the periglandular infiltration is open to debate. Perhaps there is an element of truth in both theories. Under the microscope, the picture is that of many cysts.

Degenerative.—The term degeneration of the antrum mucous membrane has crept into our rhinologic literature but the term is vague. True, degenerative changes of the antral mucous membrane, in my experience, are rare. I have found but three such specimens. The term should be enforced by a descriptive adverb.

I am not convinced that the epithelial cells in chronic inflammation are easily destroyed. I do not believe that the loss of cilia

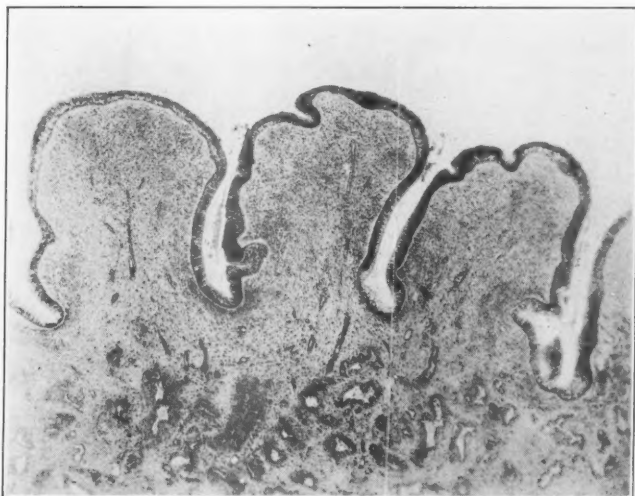


Fig. 23. Papillary hypertrophy of inferior turbinate.

with desquamation of the epithelial cells plays a prominent part in the chronic inflammations. I have been amazed to find structurally perfect ciliated epithelial cells in pus antra of long standing. My conviction has been confirmed by Doctors Mosher and Ross in their vital stain studies.

Turbinates.—The turbinate mucous membrane change is that of mucous membranes elsewhere in the nasal cavity. The same pathologic process of edema, fibrosis, infiltration and cystic formation holds true.

The terms hypertrophy and hyperplasia, as applied to the turbinates, frequently create confusion as to the pathologic process.

Hypertrophy, meaning in its truest sense, an increase in size but not in numbers of the composite parts, may be physiologic or pathologic.

An irregular papillary hypertrophy frequently occurs along the inferior margin of the inferior turbinate or over the posterior tip of the middle turbinate.

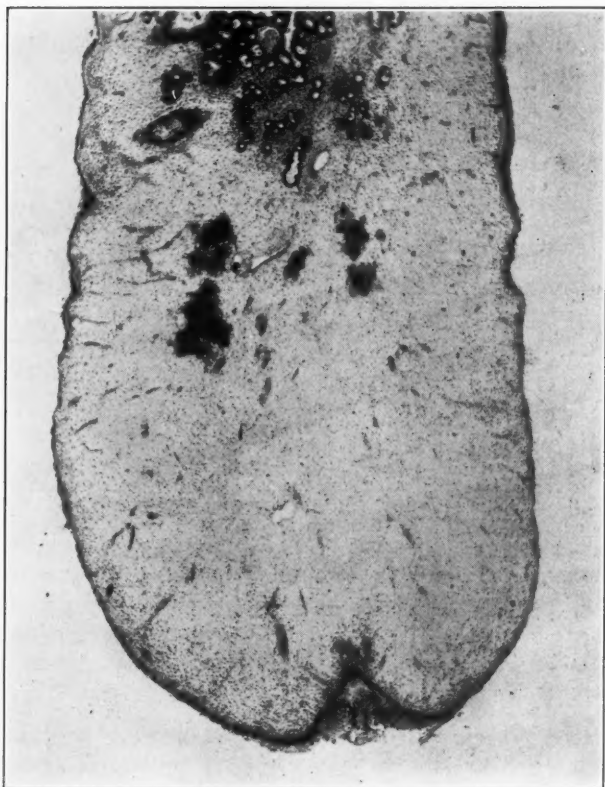


Fig. 24. Nasal polyp, 30 \times . Showing all the elements of a mucous membrane—epithelium, basement membrane and stroma containing blood vessels and glands.

Hyperplasia designates an increase in number of one or more tissue elements. In the turbinate this hyperplasia is usually of the fibrous tissue—so that the picture is that of fibrosis.

Nasal Polyps.—It is generally conceded that the etiologic factor in nasal polyps is inflammation. At one time they were considered as being myxomatous in character, but are now considered as

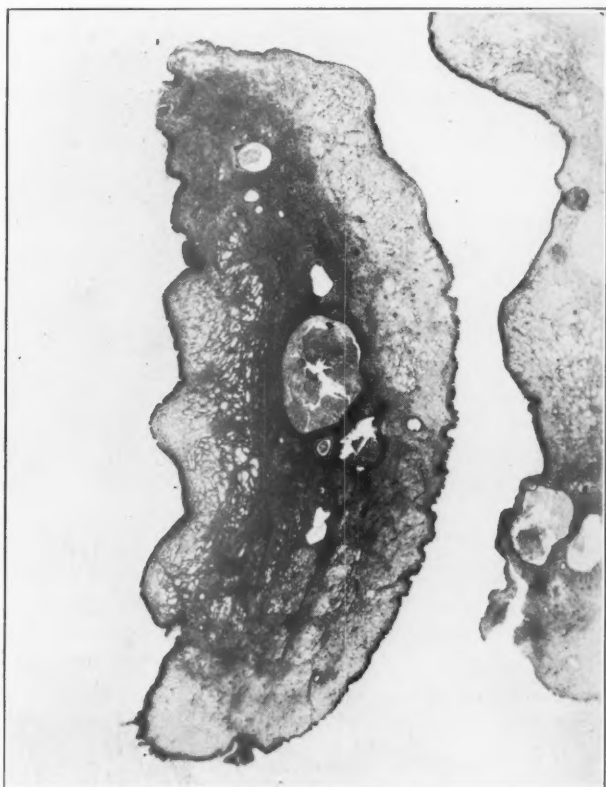


Fig. 25. Nasal polyp—cyst formation.

being of an overgrowth of tissue normal to their region of origin. In other words, they show all the elements of the mucous membrane from which they spring, and their changes are those of mucous membrane. The varieties of polyps are then (1) edematous, (2) fibrous and (3) cystic or a combination of these types.

The epithelium covering a polyp is that normal to the region in which it arose. As the polyp grows and incident to traumatization of pressure, the epithelium may become thinned to a squa-

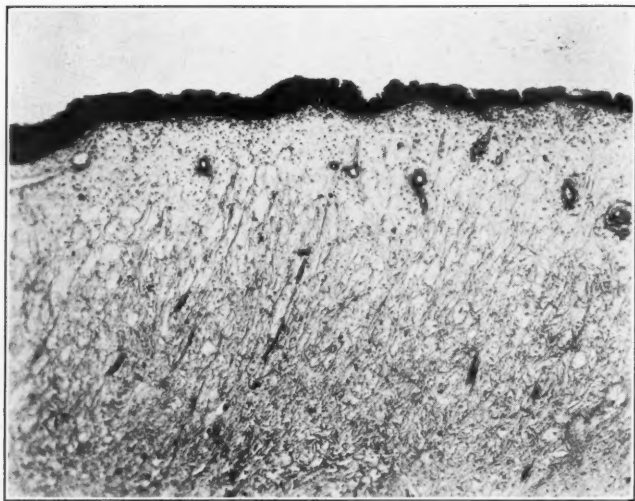


Fig. 26. Nasal polyp, 80 \times . Transition of epithelium.

mous-like layer of cells or may show true stratified squamous characteristics.

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III.

THE NASAL ACCESSORY SINUSES AS FOCI OF INFECTION IN CHRONIC ARTHRITIS.*

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The etiology of chronic arthritis is an open question, but the following statistics show very plainly that chronic arthritics often have involvement in their nasal sinuses which has given no symptoms and consequently has been overlooked. These might be termed silent sinusitides.

Drs. R. G. Snyder and S. Fineman show, out of 386 consecutive cases of arthritis, 262 cases, or 68 per cent, with pathologic changes, 1, 2 or 3 plus, in the sinus X-ray films. One hundred and twenty-six were examined by a rhinologist. Thirty-three cases (26 per cent) were negative clinically, and ninety-three cases (74 per cent), or about three out of four, were positive both clinically and by X-ray.

Of the 93 cases, 17 per cent gave a sinus history, 83 per cent were silent—that is, without subjective symptoms—and forty-two cases, or 45 per cent of the ninety-three, were not treated for various reasons, viz.: 1. No faith in sinus surgery. 2. Advised by family physician against sinus therapy. 3. Economic. 4. Other foci more important.

Of this group of forty-two cases, twenty-three, or 54 per cent plus, failed to show improvement in the arthritis, and nineteen, or 45 per cent plus, improved under general treatment. Five patients did not cooperate or finish treatment. All failed to show improvement.

Twenty-eight cases had conservative medical treatment; one no improvement; seven moderate improvement; ten marked improvement; and ten very marked improvement; the latter two groups being 71 per cent.

*Read before the American Laryngological Association, Atlantic City, May 21, 1932.

Eighteen cases that had sinus surgery showed: One, no improvement; one, moderate improvement; five, marked improvement, and eleven, very marked improvement; the latter two groups being 89 per cent.

386 CONSECUTIVE CASES OF ARTHRITIS.

262 or 68% showed pathology in the nasal accessory sinuses by X-ray.
126 of the 262 cases were examined by a rhinologist.

of the { 33 or 26% were negative clinically.

126 cases { 93 or 74% were positive clinically as well as by X-ray.

of the { 16 or 17% gave sinus history.

93 cases { 77 or 83% were silent, that is gave no sinus history.

		42 or 45% refused sinus treatment		{ 23 or 54% unimproved under general care. 19 or 45% some improvement.	
		5 cases did not cooperate, treatment unfinished, no improvement.			
The 93 cases	28 had medical sinus treatment	{ 1 no improvement 7 moderate improvement 10 marked improvement 10 very marked improvement		{ 3½% 25% 35½%+ 35½%+ } 71%	
	18 had sinus surgery	{ 1 no improvement 1 moderate improvement 5 marked improvement 11 very marked improvement		{ 5½% 5½% 27½%+ 61½% } 89%	

The sinuses involved were mainly antra and ethmoids. The ethmoiditis was of the chronic hyperplastic type—that is, with thickening of the mucosa and bony cell walls. The antral mucosa was hyperplastic, and usually some mucopus was found.

The tonsils are considered one of the most likely foci of infection, but it is interesting and important to note that seven out of the ten patients presented had had their tonsils cleanly removed without benefit. This might lead one to believe that too much stress had been put on the tonsils, when in fact the nasal sinus was the factor that upset the patient's balance. Three out of the ten patients also had had several previous operations on their sinuses.

Purulent discharge was present in only three of the ten. One gave a history of frequent colds in the head, five rarely, and three never. Only one had headaches and that one had all of the previous symptoms—that is, purulent discharge, frequent colds and

headaches. The tonsils were out and there had been three previous sinus operations. In this case a chronic sphenoid infection which had not been detected was the source of the trouble.

Procedure of the Sinus Examination.—After obtaining the history, examine the nose anteriorly and posteriorly to note the condition of the mucosa and the presence of secretion. Then shrink the nasal mucous membrane and transilluminate. If the transillumination suggests chronic frontal or antral involvement, give particular attention to shrinkage about their orifices and again note character of the mucosa and secretion.

The middle turbinates, with the mucosa of the middle meatus, tell much about what may be going on in the ethmoids, frontals and antra. I do not believe it is possible, where there is involvement of the ethmoids, frontals or antra, to find a middle turbinate whose membrane is clear, smooth, glistening or normal in color. One of the first changes noted when the ethmoids become involved is change in character of the mucosa of the middle turbinates, especially along the lower border and at the anterior and posterior ends. It is most important to note the above.

Transillumination is a great aid and should always be used as a routine examination, but too much value should not be placed upon it, for an involved sinus may transilluminate clearly, or the reverse be true, in that a sinus may transilluminate dark, where the active process has long since subsided, where the bony walls are thick or have become thickened from a previous operation. Of course, it is of no value for the ethmoids and the sphenoids, and unfortunately the ethmoids are most frequently involved.

X-ray pictures should be made in all cases of arthritis, regardless of whether or not the patient has nasal symptoms, and they must be well made. Poor films are worthless. They should be made in four stereoscopic pairs, at proper angles. These are of great value by elaborating the clinical information, but here again previous inflammations and operations may have produced fibrous tissue that will obstruct the rays. At times, however, even good films will not always confirm the clinical findings, but will appear negative. Under these conditions the rhinologist must rely on the clinical evidence.

Two of the ten patients discussed had external antral operations; the other eight were done intranasally.

The following ten illustrative cases were under treatment for some time by Dr. R. G. Snyder, before I operated upon their sinuses:

E. O., female, aet. 31. Infectious rheumatoid arthritis.

This patient was first seen by Dr. Snyder, September 18, 1929. She complained that for six months she had had pain in the jaw and difficulty in chewing.

History: She had noticed a peculiar grating sensation at left jaw articulation, not painful at first but becoming progressively worse, until it became a dull continuous ache, and a sharp pain when eating. Frequent left frontal headaches; constipation.

Physical examination: Essentially negative except for two suspicious devitalized teeth. Tonsils had been cleanly removed eighteen months previously. There was a thick greenish yellow discharge from the cervix, and considerable spasticity of the colon. These conditions were treated by Dr. Snyder.

Patient was referred to me. My examination showed a deviated septum, hypertrophied middle turbinates on both sides. Transillumination: Frontals clear, right antrum dim and left dark. X-rays showed right antrum slightly cloudy, left very cloudy, ethmoid labyrinth slightly cloudy on both sides. Sphenoids clear. The right antrum douched negatively, needle puncture of the left gave two drams of white mucopus. I advised opening both antra, but Dr. Snyder and the patient wanted to try conservative treatment. The antrum was douched for a month, with no improvement in the rheumatic pains, and then Dr. Snyder and the patient were convinced that surgery was necessary.

Operation: October, 1929. Submucous resection. Right antrum opened intranasally and one-half dram of very thick mucus found. Left antrum also opened and thickened membrane and pus found. Right middle turbinate removed. In December, 1929, there was no secretion in the nose and both antra douched negatively.

Patient was seen again in May, 1931. Said she felt fine and was entirely free from pain.

A. B., female, aet. 63. Infectious rheumatoid arthritis. Patient first seen December 2, 1927, complaining of pain in right knee of one year's duration, and swelling for the past two months.

Past history: Inflammatory rheumatism in childhood and again three years ago. Definite hereditary tendency. Tonsils were suspicious and removal had been recommended by two doctors. Constipated for thirty-five years and used cathartics.

Physical examination: Negative except that patient had high uric acid. Routine treatment for the purpose of eliminating the uric acid only improved the arthritis to a very moderate degree.

Patient had had an operation on the left side of her nose twenty-six years previously, no trouble since until the past few days. No nasal obstruction or discharge, colds rarely, occasional slight sore throat. Pro-

gressive deafness, loss right ear 76%, left 30%. Had had ear treatments for some time, at home. Teeth negative.

Physical examination: Transillumination; frontals clear, right antrum clear, left dark, punctured and douched, gave up three drams of pus. Left ethmoids bulging, mucosa soft, suspicious of infection. In the X-rays the left frontal, ethmoids and antrum all showed a marked and uniform degree of cloudiness. Left middle turbinate enlarged.

Operation: January, 1928. Left ethmoidal labyrinth opened, bony walls very thick, proliferating osteitis, no pus. Left antrum opened intranasally, thickened membrane with pus.

One year later she had no pain or disability in her joints. Audiogram showed a loss in hearing of 30% right and 33% left.

M. K., female, aet. 29. Infectious rheumatoid arthritis. Arthritis started in 1918, and became much worse following a fall in 1922. Patient first saw Dr. Snyder in 1928. She was anemic and undernourished. Bile and indican were present in the urine and there was some pathology in the colon. Treatment with colonic irrigations, baking and massage, vaccine and iron injections for anemia.

When patient was referred to me in November, 1928, she walked with the aid of a crutch which she had used for several years. Rarely had colds, slight sore throats occasionally, no headaches. Tonsillectomy six years previously. No cough. Teeth X-rays negative.

Physical examination: Extreme deviation of the nasal septum into the right side, with a low grade hyperplastic ethmoiditis on the left side. Transillumination clear. X-ray of the right antrum moderately cloudy, ethmoid labyrinth showed definite cloudiness of the cells on the left side.

Operation: December, 1928. Submucous resection and left ethmoidectomy. Opening into left sphenoid enlarged.

Patient was last seen in May, 1931. Walked unaided, able to return to work, looked and felt 100% better.

R. A., female, aet. 41. Infectious rheumatoid arthritis. Patient was seen by Dr. Snyder September 26, 1929. Complaint of "chronic sinus trouble," painful joints and limitation of motion.

Physical examination: Essentially negative except for several devitalized teeth, but no abscesses.

Had had a left radical antrum in 1927, septum operation ten to twelve years previously, right radical antrum in February, 1928. Acute left antrum last summer, has frequent colds, considerable discharge left, and left occipital headaches. Occasional sore throat, tonsils out in 1906.

Physical examination: Straight septum, middle turbinates out. Transillumination: Frontals clear, right antrum very dim, left almost clear. Right antrum douched negatively, left, one-half dram of mucopus. Ethmoids partially out on the left side. Left sphenoid full of mucopus. X-rays showed both frontals clear, ethmoids right slightly cloudy, left moderately cloudy. Both antra diminished in size and moderately cloudy. Left sphenoid moderately cloudy.

Operation: October 9, 1929, under local anesthesia, much scar tissue removed from in front of the left sphenoid, posterior part of the nasal septum had been removed. Left sphenoid entered, anterior wall very

hard and thick, contained pus. Sphenoethmoidal wall removed external to the sphenoid, and some ethmoidal cells along the orbit. Polypoid tissue in antrum around the natural orifice removed.

Since discharge the patient has been perfectly well.

C. J. H., female, aet. 50. Infectious rheumatoid arthritis. November 30, 1925, patient reported to Dr. Snyder, complaining of pain in knees, back and fingers, of five years duration. Definite hereditary history. History and physical examination essentially negative except for occasional attacks of indigestion.

In 1920, I removed this patient's tonsils. Saw her in December, 1925, complaining of pain in right cheek for three days. Right antrum transilluminated dark, left clear. Douche through the natural orifice produced two drams of mucopus. On January 8, antrum douched clear. May, 1926, patient had another flareup of the right antrum, transillumination clear, but the X-rays showed it to be cloudy. Stereoscopic films in the lateral position showed a breaking down of the ethmoid cells septa, right, anteriorly, and a cloudiness of the posterior cells.

Operation: May 28, 1926, right ethmoids cleaned out, found very hard bony walls with thickened mucosa. Middle turbinate had been removed. Right antrum opened under the inferior turbinate, mucosa found to be thickened. Enlarged right sphenoidal orifice. Patient recovered completely and has been well ever since.

H. A. S., female, aet. 43. Osteo-arthritis. Patient first seen by Dr. Snyder, May 16, 1928, complaining of pain in the right knee, shoulders, wrists and joints of hands, duration fourteen years.

Past history: Scarlet fever in childhood followed by acute rheumatic fever which seemed to localize in the knees and joints of the arms, acutely painful. Also had measles and mumps. Operation on antrum. Tonsils and adenoids had been removed.

Physical examination: X-ray revealed marked arthritic changes of hands and knees. Marked absorption of the alveolar border around the remaining teeth. X-ray of colon showed a moderate residue after evacuation. Treated with colonic irrigations.

Patient was referred to me in 1928. In 1919 she had had a left antrum operation, twice in 1920 had had hole in antrum enlarged, also had had septal operation. Never had had pain in nose or foul odor. The anterior half of the right inferior and middle turbinates had been removed. A hole into the right antrum under the inferior turbinate through which could be seen mucopus and thickened mucous membrane. Mucopus came from the right posterior ethmoidal cells. X-ray showed the frontal and ethmoidal sinuses clear on both sides. Sphenoids looked clear in the lateral films. Left antrum clear, right cloudy with thickened membrane.

Operation: September, 1928, under local anesthesia, a radical operation was performed on the right antrum. Some polypoid tissue was found superiorly and inferiorly, with a bony partition in the anterior floor and much degeneration of the mucous membrane in front of it. November, 1928, operation on right ethmoids; thickened bony walls, polypoid mucous membrane and mucopus in the cells were found.

No improvement of the arthritis.

L. S. A., female, aet. 59. Mixed arthritis. First seen January 15, 1931, complaining of pain and stiffness of knees, hands and feet of five years' duration. She had walked with crutches for four years.

History: Tuberculosis of the hip at the age of twelve. Sciatica of the right leg. Father had rheumatism badly.

X-ray of the colon showed marked atonicity, and a large residue after evacuation. Both knees showed marked arthritic and atrophic changes in the bones, and marked destructive changes of the cartilages.

Patient came to my office on crutches. She had colds and headaches rarely, no cough.

Physical examination: Atrophic nasal mucosa with crusts. Transillumination clear except for the left antrum which was dim, douching yielded four drams of pus. Small flat tonsils, no debris. Sinus X-rays showed a fluid level in the left antrum, left ethmoids and right sphenoids cloudy.

Operation: February 19, 1931. Under local anesthesia, a radical right antrum operation was performed. We found thickened mucosa with free pus.

April 14, 1931. Patient departed for home, no longer using crutches. Improved.

L. A. D., female, aet. 53. Rheumatoid arthritis. December 7, 1925, this patient complained of rheumatism in her neck, hands, toes, knees and lower spine of three years' duration.

History: Patient had had rheumatism in her right arm for a few days when in her early twenties. Had been constipated for ten years.

X-ray of the colon revealed considerable fecal matter throughout the bowel and a large residue after evacuation. Patient was treated by Dr. Snyder with tolysin, colonic irrigations, thyroid and vaccine.

She complained of occasional colds, no sore throat and no nasal discharge.

Examination: The right antrum transilluminated dark and on douching yielded four drams of foul-smelling pus. X-ray, frontal sinuses absent. The ethmoids and sphenoids were clear, the right antrum was definitely cloudy. Patient had three dead teeth which were not in relation to the right antrum.

Operation: In December, 1925, a radical operation was performed on the right antrum, under local anesthesia. The antrum healed and has been entirely negative since. Arthritis cured.

J. C. S., female, aet. 76. Osteo-arthritis. This patient had had osteo-arthritis for twelve years, mainly in the knees. Nose dry with some post-nasal discharge. Colds rarely, no headaches, sore throat or cough. There was some debris in the left tonsil.

Examination: Transillumination; left antrum slightly dim, right dim. Sinus X-rays showed a fluid level in the right antrum (3+). Left negative. Right antrum puncture 1+ mucopus.

Operation: An intranasal right antrum operation was performed, and soft, edematous, polypoid mucosa found.

Report received one year later states that the arthritis is markedly improved.

W. F. D., male, aet. 22. Mixed arthritis. Patient had had arthritis, mainly of the spine, for eight years. Had an infected prostate which was not gonorrheal.

He complained of no nasal trouble, and said he had colds, headaches or sore throat rarely. Tonsils had been operated upon twice.

Physical examination: There was a very marked septal deviation to the right which obstructed all view of the right middle turbinate and ethmoids. The left middle turbinate was boggy suggesting slight trouble on that side. There was a small adenoid and tonsil remnant.

X-ray of the sinuses showed the right ethmoids to be 2+, and the left 1+.

Operation: Submucous resection of the nasal septum. We found thickened membrane in the right ethmoids, but the left ethmoids looked normal after removal of the middle turbinate. Adenoid remnant was removed.

Five months later he reported that the arthritis was much improved.

CONCLUSIONS.

1. That nasal sinus involvement is an important factor in chronic arthritis.
2. That the sinus involvement often gives few or no symptoms and might be termed "silent" sinusitis.
3. That infection of the tonsils, in this short series of cases, is not as important as the sinusitis.
4. That most of the cases have no frank, purulent nasal discharge.
5. That best results are obtained in sinus infections by surgical measures.

39 EAST FIFTIETH STREET.

IV.

PULMONARY COMPLICATIONS OF TONSILLECTOMY WITH REPORT OF THREE CASES.*

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PHILADELPHIA.

Hemorrhage, anesthetic shock and pulmonary involvement are the three grave complications which may follow the operation for the removal of tonsils and adenoids. Hemorrhage is usually uppermost in the operator's mind, for no operation, even electro-coagulation, can be termed entirely bloodless. Anesthetic shock has led to extensive status lymphaticus and thymus studies by the British Medical Society and in various parts of our own country. Its endocrine significance was reviewed before the State Society by Campbell in 1931.

An extensive study of pulmonary abscesses following tonsillectomy based upon reports from questionnaires sent to 1,020 laryngologists in the United States and Canada, with analysis of about 200 positive cases, an incidence of one in each 2,500 to 3,000 cases, was published by Moore in 1922. This small ratio would naturally be accepted by most operators with a tranquil mind. It would be natural to assume that this occasional accident had occurred through some carelessness in routine or predisposing pathology. Yet this has happened to us—after thirty years of active service in connection with the tonsil operation in several hospitals, with scarcely a case of definite pulmonary sequence—three cases have occurred in services under the writer's direction, two of lung abscess and one of pneumonia, in the past two and one-half months, and these have followed what were reported as perfectly normal tonsillectomy and adenoidectomy operations.

It is obvious that these should be reported as a matter of record. Operations were done in each case after routine reference from

*Read before the Section on Otolaryngology of the College of Physicians, Philadelphia, October 19, 1932.

the outpatient department, with the customary preliminary safeguards performed in each case by a different operator, under ether anesthesia, in the prone position and with the use of suction tube.

Case 1.—C. M., a boy, aged 3, was admitted to the Children's Hospital August 8, 1932, for tonsil and adenoid operation, and sent home the following day. On August 11, 1932, was brought to the medical dispensary with fever of 103° . Child was examined and prescription given. Four days later he returned to the nose and throat dispensary. Throat was about normal, but the left ear was injected. Four days later he returned again, the left tympanic membrane was incised, right was noted as dull, and he was admitted to the hospital with a temperature of 100° . Both tympanic membranes were incised August 20, 1932. Temperature continued to fluctuate between 100 and 103 . Blood studies showed R. B. C. just below 4,000,000, W. B. C. 11,200 and 10,300, and polynuclears 72 and 70 per cent. Patient developed a cough and foul breath. Mastoid and sinus X-ray studies were negative. Sputum examination showed mostly hemolytic streptococci.

Chest X-ray revealed a large dense area filling the right upper lobe and a circular layer within this reaching from the second to the fourth rib, due either to abscess or cavity. Medical consultation found bronchovesicular breathing with occasional râles in the right chest anteriorly. Case was referred to the University Hospital for bronchoscopic drainage. Culture of bronchial secretion showed predominance of hemolytic pneumococci with a moderate amount of nonhemolytic streptococci, and other strains.

Biweekly bronchoscopic treatment and postural drainage were given by Dr. Tucker. Left mastoid was suspicious but both ears have cleared and the child is practically well.

Case 2.—E. D., a girl of 5, was operated on at the Mary Drexel Hospital for hypertrophied and diseased tonsils September 8, 1932. Slight bleeding from the left tonsil required one suture. The day following operation the temperature rose to 102° . This continued irregular for a week, from 100 to 101 degrees, then went to 104° five days later. Crisis occurred the next day. The child coughed up much mucus and the temperature gradually dropped to normal. Nose, throat and ears appeared negative the

first week. On the ninth day fine râles were heard at the left base and the next day diagnosis of bronchopneumonia was made. X-ray examination showed marked infiltration at the roots of both lungs, clouding of the left lower and lower half of the left upper lobe. No cough or other subjective symptom was noted until the ninth day. Blood examination was conventional with the highest W. B. C. 16,000. Sputum examination showed, among other flora, a few fusiform bacilli and spirilla. Final X-ray on September 30, 1932, showed thickened plura and some infiltration at the root of the left lung but no consolidation or fluid. Child was discharged on September 30, 1932, in good condition.

Case 3.—C. A., a girl of 3, with chronic nasal discharge and septic tonsils, was admitted to the Children's Hospital and tonsils and adenoids were removed September 9, 1932. She was seen three days later in the nose and throat dispensary and the throat was healing well. She was brought to the medical dispensary two days later with a temperature of 101°. Two days later she was admitted to the hospital with a temperature of 100.8°. The chest examination showed slight limitation of respiration and increased tactile fremitus over the right upper lobe, impaired resonance with coarse bronchial râles over both sides. For a few days chest signs increased, with difficulty in coughing up mucus, but temperature remained about 102.

First X-ray on September 15, 1932, showed an area of mottled density with poorly defined borders in the fifth extending to the sixth interspace posteriorly on the right side. This was diagnosed as lobar pneumonia or beginning lung abscess. Diaphragm on the right side was elevated, but heart and trachea were in normal position. An X-ray the next day showed widespread, finely mottled infiltration, scattered throughout the right lung and to a lesser extent in the left. This suggested tuberculosis. An X-ray four days later showed infiltration scattered throughout both lungs—more marked in the right. Diagnosis was in doubt between tuberculosis, bronchopneumonia and multiple small abscesses. The child has been improving under treatment and a later X-ray, October 4, 1932, showed diminution of the mottled area. The

condition presented the initial flare of, if not actually, pulmonary abscess with a tubercular background. Bronchoscopic drainage has been delayed.

COMMENT.

Two of these children have recovered and the third is probably retarded by pre-existing tuberculous involvement.

In the Moore Survey, 60 per cent of the cases were reported with involvement of the lower lobes. In Case 1, the abscess occurred in the right upper lobe; in Cases 2 and 3 infiltration was present in the left upper but more marked in the left lower lobe.

The incidence of fusiform bacilli and spirilla of the Vincent's organism in Case 2 may have been significant and bear some relation to the suppression of frank pneumonic signs.

1912 SPRUCE ST.

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V.

THE CAVERNOUS SINUS: ANATOMICAL AND CLINICAL CONSIDERATIONS.*

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PHILADELPHIA.

In the consideration of certain medical and surgical conditions the greater interest lying in the clinical picture and therapeutic efforts may lead us to underestimate or overlook anatomic peculiarities which when thoroughly understood and properly evaluated are apt to change our ideas of the etiology and pathology of these conditions. Such a situation is perhaps exemplified in the distressing condition of cavernous sinus thrombophlebitis in which the emphasis has usually been placed on the clinical and therapeutic sides with but little consideration of anatomic features that make this condition of much interest, especially from the etiologic and pathologic standpoints. Without perhaps adding anything new to the anatomy of this particular region, I wish to review certain features and emphasize certain peculiarities that may make more clear to us the development of cavernous sinus thrombophlebitis. My aim will be to discuss but briefly the clinical side of the picture and to emphasize particularly the anatomic peculiarities of the sinus itself and the venous drainage of the head and face which have such an important bearing on the development of thrombosis of this blood channel. To bring out these points it will be necessary to mention in some detail the venous connections and the anatomy of the cavernous sinus.

VENOUS CONNECTIONS OF THE CAVERNOUS SINUS.

The cavernous sinus, situated one at each side of the body of the sphenoid bone, has rather widespread venous connections. They receive venous blood:

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From the Department of Otolaryngology of the University of Pennsylvania.

1. From the dura mater through the sphenoparietal sinus.
2. From the cerebrum and meninges through the inferior and anterior cerebral and middle meningeal veins.
3. From the mucous membrane of the sphenoid air cavity through small veins penetrating the bony wall of this nasal accessory sinus.
4. From the forehead, supra-orbital region, nose, eye and other parts of the upper face, through the main venous supply—the superior and inferior ophthalmic veins and their tributaries.

The frontal and supra-orbital veins unite at the root of the nose to form the angular vein, which courses downward and at the lower margin of the orbit becomes the facial vein. Branching off from the angular is a small tributary called the nasal-frontal branch, which communicates with the superior ophthalmic vein. As the superior ophthalmic continues toward the cavernous sinus it receives the following: At the margin of the orbit, a tributary from the frontal diploe; within the orbit, the anterior and posterior ethmoid veins from the ethmoid and sphenoid air sinuses and the intranasal tissues; and the ciliary veins from the tissues of the orbit. It eventually enters the anterior end of the cavernous sinus at the sphenoid fissure. The inferior ophthalmic vein originates in a network of small veins on the inner portion of the floor of the orbit. This plexus communicates with the facial veins and continues backward toward the fundus of the orbit, receiving branches from the various eye muscles. It opens either directly into the cavernous sinus or else unites with the superior ophthalmic vein just before entering the sinus. An important branch of the inferior ophthalmic communicates with the pterygoid plexus through the sphenomaxillary fissure.

Branches leading blood away from the cavernous sinuses are as follows:

1. Small veins that pass from its under surface through the foramen ovale, the canal of Vesalius, the foramen lacerum medium and the foramen rotundum to communicate with the pterygoid plexus. Through these branches there exists an indirect relationship between the cavernous sinus and the soft palate, the fauces, pharynx and alveolar processes, the veins from these areas enter-

ing the pharyngeal and pterygoid plexuses which communicate with each other.

2. A number of fine veins which accompany and surround the internal carotid artery in its course through the carotid canal and thus reach the jugular bulb.

3. The superior and inferior petrosal sinuses, which carry the blood from the posterior extremity of the cavernous sinus into the lateral sinus.

In addition to the branches leading to and from the cavernous sinus there are intercommunicating branches which serve to equalize the venous pressure between the two cavernous sinuses. They are the following:

1. The coronary or circular sinus, consisting of a double cross anastomosis, between the cavernous sinuses, lying in front of and behind the infundibulum of the pituitary body.

2. The transverse occipital or basilar sinus at the caudal end of the cavernous sinuses, serving as an intercommunication between them.

While part of the blood entering the angular vein is led off into the superior ophthalmic, the greater portion is carried on into the facial vein, which continues its course downward and backward through the cheek, receiving branches from the eyelids, alæ and tip of the nose, the lips, facial muscles, parotid gland, the tonsils and submaxillary glands. At the level of the hyoid bone the facial vein enters the internal jugular. An important communication of the facial vein is the branch known as the deep facial or anterior internal maxillary vein originating in the pterygoid plexus and opening into the facial vein where that vessel passes beneath the zygomatic muscles.

This pterygoid plexus is embedded in the adipose tissue occupying the pterygoid fossa around and between the pterygoid muscles. As has been mentioned above, this plexus communicates with the facial vein through the deep facial branch; with the pharyngeal plexus by anastomatic branches; with the cavernous sinus through the small veins which traverse the foramina ovale, Vesalii, lacerum medium and rotundum; and with the inferior

ophthalmic vein through a small branch passing down through the sphenomaxillary fissure.

It can be thus seen there is here a circle of vascular channels intimately connecting the peripheral tissues of the face, head and throat with the large venous channels of the brain. The normal flow of blood from the frontal and supra-orbital veins is both down through the angular into the facial vein and also across from the angular into the superior ophthalmic and on into the cavernous sinus. The blood in the tributaries of the ophthalmic veins draining part of the interior of the nose, the ethmoid, sphenoid and frontal sinuses should flow into the cavernous sinus except the small part that passes down the small branch from the inferior ophthalmic to the pterygoid plexus. The branches of the facial vein draining the external nose, the lips and face should normally flow down the facial vein into the internal jugular. The pterygoid plexus receiving blood from the pharyngeal, faucial and dental regions should transmit its blood either anteriorly into the facial vein through the deep facial branch or posteriorly into the temporomaxillary vein which unites with the posterior auricular to form the external jugular vein.

REGULATION OF THE VENOUS BLOOD IN THE CRANIUM.

In establishing connections of outlying areas to the cavernous sinus it is important to take into consideration the fact that the facial, angular and ophthalmic veins are destitute of valves or contain such rudimentary valves as to form no bar to the passage of blood in a reverse direction. Moreover, all the intracranial veins, as well as those of the pterygoid plexus and the small veins joining it with the cavernous sinus are free of all valves and allow free flow of blood in either direction. As a matter of fact, the direction of flow in these intracranial venous channels and the emissary veins is frequently changed, due to such conditions as changes of gravity, muscular exertion, the relative pressure within and without the cranium and the respiratory cycle.

The latter factor alone is of great importance in regulating this flow of blood. On inspiration there is a rapid increase in the rate of flow of the blood in the internal jugular vein into the large veins of the chest. This has a tendency to draw the blood rapidly

through the large venous sinuses, but this is partly compensated for by the following factors which tend to retard the flow of blood through these channels:

(1) The oblique entrance into the longitudinal sinuses of its tributaries—the larger middle and posterior cerebral veins pouring their blood into it against the stream; (2) the division of the blood at the torcular Herophili into two trunks diverging at right angles; (3) the course of the blood current in the lateral sinus—first horizontal, then vertical in the first part of the sigmoid, then horizontal again and finally a quick upward and outward turn with considerable narrowing at the jugular foramen before emptying into the internal jugular vein; and (4) the presence of the numerous interlacing bands of fibrous tissue in the longitudinal and cavernous sinuses which considerably narrow these vessels and tend to retard the current of blood.

However, while these factors operate to some extent in slowing the flow of blood through the intracranial channels during inspiration and prevent too great an aspiration of blood from the brain there is no doubt some aspirating effect reaching as far as the emissary vessels of the cavernous sinus, the chief of which are the ophthalmic veins. This aspirating effect would tend to hasten the flow of blood from the ophthalmics into the cavernous sinus and at the same time draw blood from the angular at such a rate that at times the flow in the facial vein would be reversed into the angular and ophthalmics. Furthermore, the aspirating effect would tend to draw blood from the pterygoid plexus up the small valveless emissary veins to the cavernous sinus.

The opposite to this aspiratory effect would be experienced in the expiratory phase of respiration and in any muscular exertion or strain at which time there is great distention of the internal jugular vein with a tendency to a reversed current of blood through the jugulars and the large intracranial sinuses. Such a reversed current, if sufficiently strong, would be transmitted to the cavernous sinus and out into the facial vein through the ophthalmics.

In examining the venous flow into and out of the cranium we must not overlook the fact that the aspiration of venous blood

on inspiration, and the reverse on expiration or muscular strain is transmitted from the internal jugular to the facial vein as well as through the larger intracranial venous channels. If this force were transmitted equally to the facial and venous sinuses there should be no change in the current of blood through the cavernous sinus and its emissary veins. However, in all probability, such forces are not distributed equally to these different channels and the result would be then a frequently changing direction and rate of flow through the intracranial sinuses and the facial vein and its tributaries. Therefore, it can be assumed that blood may flow through the petrosal and cavernous sinuses out into the ophthalmic at times of severe muscular strain and may flow up the facial and emissary veins of the cavernous sinus from the pterygoid plexus, depending on the relative difference in the forces exerted on different parts of this venous circle.

DETAILED ANATOMY OF THE CAVERNOUS SINUS.

Before applying these facts to the occurrence of thrombosis of the cavernous sinus let us go, in some detail, into the anatomy of this sinus itself.

The cavernous sinuses, situated one on each side of the sella turcica, measure about 2 cm. in length and have a diameter of about 1 cm. They are contained within the reduplications of the dura mater which form the lateral walls of the osseomembranous pituitary fossa and are directed cephalocaudally extending from the sphenoid fissure in front to the apex of the petrosa behind, where they terminate by dividing into the superior and inferior petrosal sinuses. In a considerable part of their course they overlap slightly the roof of the sphenoid cavity and are separated from this air cavity by the thinnest of bony partitions. The lumen of the cavernous sinus is greatly reduced in size by being traversed by numerous trabeculae, a sympathetic plexus, the large internal carotid artery and the abducens nerve. The artery enters the sinus, running first forward and upward and then by turning directly upward pierces the roof of the sinus to emerge into the cranial cavity. The abducens nerve enters the cavity of the sinus, passing from behind forward and from above downward toward the sphenoid fissure.

In the outer wall of the cavernous sinus, situated from above downward, are the oculomotor, the trochlear and the ophthalmic and maxillary divisions of the trigeminus nerve, passing forward on their way to the sphenoid fissure and foramen rotundum by means of which they enter the orbital cavity. The ophthalmic division enters the ventral portion of the outer wall of the sinus as the sinus passes over the mesial portion of the Gasserian ganglion. The trochlear nerve lies just above the ophthalmic and follows a slightly oblique course through this wall of the sinus and upon arriving at the sphenoid fissure is slightly dorsal to the oculomotor. The latter nerve enters the outer wall of the sinus in a manner similar to that followed by the trochlear nerve, but is placed more dorsally and passes close by and ventral to the posterior clinoid process. Its direction is slightly downward so that in its course forward it passes slightly mesial to the trochlear and appears slightly below this nerve at the sphenoid fissure. The ophthalmic and oculomotor nerves divide into their branches in the anterior one-fifth of the sinus.

The main feature I wish to emphasize in the anatomy of the cavernous sinus is the narrowing of its lumen by the network of trabeculae traversing it. While textbooks and articles referring to its anatomy all mention this network, the denseness and extensiveness of these trabeculae have perhaps not been sufficiently emphasized, for they play an important part in thrombophlebitis of this sinus. Examination of many specimens of the cavernous sinus, both in cross section and in longitudinal division, has impressed upon me the obstructive nature of these bands of tissue. Winslow, the anatomist, was struck by this peculiar arrangement, noting that the cross section had much the appearance of a section of the corpus cavernosum penis and led him to give the sinus its name. There appears to be no set order in the placement of these trabeculae across the lumen of the sinus. They are formed of fibrous tissue and vary in size from the tiniest thread to a diameter equal to or greater than the trochlear nerve. They cross from side to side, diagonally and more or less longitudinally in the sinus and from them, and from the walls of the sinus fringe-like prolongations, 0.5 to 2 mm. in length, project freely into the lumen. These bands are attached not only to the walls of the

sinus but also to the two structures running through its lumen, the internal carotid artery and the abducens nerve. These fibrous bands are rather peculiar in that they are all coated with a lining membrane of endothelium similar to that lining the inner wall of the sinus itself. The walls being formed of dura mater lack any extensive development of elastic fibers and therefore are of more rigidity than extracranial veins.

It is the number of these criss-cross trabeculae in the sinus that is surprising, for they appear to be so extensive that its use as a vessel for the transmission of blood appears to be in danger of being lost. In places the bands are so thick and interlaced that crevices and pockets are formed which would probably filter out the corpuscles and even block the flow of blood.

Without doubt Nature has provided such an arrangement to serve some useful function, but what that function is appears to be somewhat obscure. It would seem that the most acceptable explanation is that this obstructive arrangement in the sinus is part of the system previously mentioned of slowing the circulation of venous blood through the brain and preventing its too rapid depletion during the respiratory phase and the effects on the circulation of muscular strain. In the proper nourishment of the brain by means of the arterial supply and the establishment and maintenance of the circulation of cerebrospinal fluid it is necessary that the circulation of blood through the brain be slow. If the blood were drawn rapidly through the venous sinuses perhaps the flow through the arterioles into the venous channels would be too rapid to allow of sufficient nourishment of the brain substance. Furthermore, it is necessary for the blood flow to be reasonably slow in the choroid plexus, so that an osmosis will occur into the ventricles thereby originating the cerebrospinal fluid. Again it would seem that the blood flow in the large venous sinuses must be slow with but little pressure present so that the Pacchionian bodies located in and around these sinuses can perform their function of passing on the cerebrospinal fluid back into the venous sinuses, thereby completing the cerebrospinal circulation.

It appears, therefore, that while the tortuosity of the lateral sinus and its narrowing at the jugular foramen tend to slow the

blood current and prevent too rapid depletion of intracranial blood at that exit to the intracranial circulation, the cavernous sinus with its lumen so obstructed tends to do the same thing at the other end, serving as a partial block to the blood entering it from the ophthalmic veins, or, when the current is reversed, serving as a means of retarding the too rapid exit of blood from the cranium.

While this arrangement of the cavernous sinus appears to be admirable for this purpose, it constitutes a source of danger from the standpoint of thrombosis. The construction of all the sinuses predisposes them to thrombosis, their rigidity, their width, the slow circulation and, in the case of the cavernous sinus, the interlacing fibrous network of its lumen, being the important factors. It might be supposed that because of its anatomic peculiarity the cavernous sinus should become thrombosed more frequently than the lateral sinus. Such would very likely be the case if pathologic conditions adjacent to the cavernous sinus were of the character of those near to the lateral sinus. Although the sphenoid air sinus is very close to the cavernous sinus, and infection of it is very common, this infection does not develop very often into a necrosis of the bony walls of the accessory sinus and an involvement of the veins leading to the cavernous sinus. On the other hand, an infection of the mastoid air cells is of such a character as to frequently cause necrosis of the intercellular bony framework with occasional thrombophlebitis of its veins which can extend into the lateral sinus. Furthermore, it is gradually becoming apparent that thrombosis of the cavernous sinus is a much more common occurrence than has been represented in the past by the number of cases reported.

It is important here for us to distinguish between a thrombosis of the cavernous sinus and thrombophlebitis. The addition of the phlebitis to the thrombosis makes a tremendous difference. The formation of a thrombus in any vein is a protective reaction and should cause no symptoms or complications if the collateral circulation is adequate and if this thrombus does not become infected. A sterile thrombus of the cavernous sinus completely blocking its lumen may therefore cause no symptoms if it is confined to this sinus alone. There should be no edema, ecchymosis or proptosis

of the eye, because the venous blood from the eyes can flow from the ophthalmic reversely into the angular and facial veins. There should be no paralysis of the oculomotor, trochlear or first and second division of the fifth, because these nerves are embedded in the dural wall of the sinus, and should not be affected if there is no inflammatory condition present. There should also be no paralysis of the abducens, because an uninfected thrombus should not make sufficient pressure on this nerve to paralyze it.

An infected thrombus (thrombophlebitis) of this sinus, however, gives marked and rapidly advancing symptoms. The thrombosis spreads rapidly to the communicating veins in its attempt to prevent the infection from entering the circulation. There is soon involvement of the ophthalmic veins, causing the characteristic eye symptoms. The infection in the cavernous sinus soon involves the abducens nerve, causing an external rectus paralysis. Then follows rapidly involvement of the nerves in the sinus wall from extension of the infection, and very soon there develops a meningitis, due, as *Eagleton pointed out, to an associated phlebitis of the inferior cerebral veins which run into the cavernous sinus. As these veins run free without fibrous sheath in the fluid of the large lake of the chiasmal cisterna the infection is rapidly spread through the basal cisterna and over the cortex.

The primary sources of infection by which such a thrombosis or thrombophlebitis of the cavernous sinus may occur, are too well known to require any repetition here. I should like only to mention one or two features concerning the pathways such infections traverse, with emphasis being placed on the importance of the reticulated arrangement in the lumen of the cavernous sinus in the development of such conditions. In the great majority of such cases the extension to the cavernous sinus is accounted for by a retrograde thrombophlebitis beginning at the primary focus of infection, but such an explanation will not satisfactorily explain the rapidity with which cavernous sinus symptoms appear following the original infection. For instance, there are some cases of infection of the lip, nose or tonsils which are followed in a very short time (one to three days) by symptoms typical of in-

*Eagleton, W. P., *Arch. of Otolar.*, 1929, 10:653.

fection in the cavernous sinus (those referable to involvement of the third, fourth and sixth nerves). To have symptoms representing paralysis of these nerves, we must assume that there is an infection present in the cavernous sinus of such severity as to extend into the venous wall and involve these nerves. When the infection once lodges in the cavernous sinus such symptoms may develop rapidly, but the infection can reach the cavernous sinus so rapidly only by an infected embolus lodging in the intrasinus network from the original focus of infection and not by a retrograde thrombophlebitis.

We must not lose sight of the pathology in the development of thrombosis and thrombophlebitis. This pathology has to do with the struggle that nature is making to limit and control the infection. There is first the attempt to prevent the infection from reaching the circulation by a sterile thrombosis of the small veins immediately around the infection. If the infection is quickly controlled such a thrombus does not become infected and does not spread to any great extent in the veins. However, if the abscess does not receive external drainage the thrombus soon becomes infected, and it is then that extension of the thrombus occurs, for nature still attempts to prevent this infected material from reaching the blood circulation by extending the thrombus ahead of the infection. If the infection is not drained at its source there is then a tendency for it to involve the blood clot with a breaking down of its elements and the formation of pus. However, this breaking down process with extension of the infection along the vein is a comparatively slow process, depending considerably on the virulence of the causative organism. If the infection in the vein overtakes the clot formation ahead of it, then there develops the chill and high fever coincident with the throwing into the general circulation of this infected material. It is these emboli that are chiefly concerned, I believe, in the sudden development of cavernous sinus thrombosis.

In the development of a thrombophlebitis in the face and head there are important considerations to be studied in regard to anatomic conditions present that have previously been mentioned. Of first importance is the fact that the facial veins, all the intracranial veins and those emissary veins of the cavernous sinus,

including the ophthalmic and the veins to the pterygoid plexus, are devoid of valves. An additional important factor is the slowness of the circulation in these veins with a tendency frequently for a reversal of the blood current.

As an example of the probably typical development of cavernous sinus thrombosis, let us take a case with primary infection of the lip. There develops around this infection a sterile thrombosis of small veins. The infection spreads, involves the thrombus in a vein, extends in this labial vein toward the facial, the sterile thrombus forming just ahead of the infection. As the thrombotic process with the infection close behind reaches the larger facial vein, let us suppose there is some sudden muscular exertion, a fit of coughing, vomiting or other disturbance. The damming back of the blood in the internal jugular vein causes a reversed current up through the facial vein and coincident dilatation of the vein. This may easily be sufficient to break off pieces of the infected clot and carry them up through the angular vein into the ophthalmic and from there into the cavernous sinus, where they become lodged in the fibrous network of its lumen. These infected emboli are then the seat of a rapidly growing infection and thrombosis, with the current of blood ebbing and flowing through its channel hastening its development. This leads quickly to involvement of the third, fourth, fifth and sixth cranial nerves, extension to the ophthalmics with the edema, proptosis and ecchymosis characteristic of their involvement and extension to the cerebral veins with a rapidly developing meningitis.

It appears to me, from a careful study of many reported cases and from the close observation of a case of my own secondary to peritonsillar abscess following tonsillectomy, that such is the mode of infection of the cavernous sinus in the great majority of cases—that is, the dislodgment of infected particles of thrombus perhaps close to the original focus of infection and the transmission of these emboli to the meshwork in the cavernous sinus. I do not wish to imply that all cases are of this kind, for the history in some cases is typical of a gradual extension of a retrograde thrombophlebitis to the sinus, the process extending for a number of days to several weeks before the symptoms of cavernous involvement appear.

From a practical standpoint, the recognition of these principles appears to have little or no value. The infection of the cavernous sinus usually appears with such suddenness as to preclude any active attempt to prevent it. When it is established we are dependent upon certain therapeutic measures that have been advocated by Eagleton, Mosher and others, such as ligation of the carotid artery and evacuation of the sinus through the orbit, through the petrous pyramid or through the sphenoid sinus. The dangerous feature of cavernous sinus infection is the complicating meningitis which terminates these cases, and until more satisfactory therapeutic measures for combating septic meningitis have been established these cases will continue to give the extremely high mortality they now have.

2117 CHESTNUT STREET.

VI.

RESISTANCE OF THE UPPER RESPIRATORY MUCOSA TO INFECTION.

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ST. LOUIS.

Studies on the functioning of the ciliated epithelium of the respiratory tract have revealed its marvelous efficiency as a protective mechanism. The factors of greatest importance in the struggle of the mucosa against invasion are mucus secretion, ciliary action and phagocytosis. Other factors, such as local immunity, local reaction and lysozyme, play a part under certain conditions. No general agreement is found as to the relative importance of these factors. It is most logical, however, to recognize that mucus secretion, ciliary action and phagocytosis are interdependent and supplementary in their actions. Mucus secretion is necessary for the surface activity of both cilia and phagocytes. Ciliary action keeps the viscous film of mucus moving toward the pharynx, thus dooming to destruction all but a very small proportion of potential infections. The necessity for phagocytic activity is in this way confined largely to those cases where the function of the mucociliary layer has failed.

It is very difficult for anyone who has observed the vigorous brush-like activity of cilia to understand how even the most vigorous of motile bacteria could penetrate such a barrier. Furthermore, the viscosity of the mucus furnishes high resistance to penetration. These facts lead one to the conclusion that in order for penetration of the ciliated mucous membrane to take place, there must be an alteration in the nature of the mucus secretion or an area of stagnation present or produced by the infecting micro-organism. Rejection of this hypothesis necessitates acceptance of the quite improbable idea that bacteria are intermixed with the mucus and penetrate the normal membrane which is protected by normal mucus secretion and normal ciliary action.

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Stagnation when already present is a distinct aid to infection and may be brought about by an anatomic defect, alteration of the mucus secretion, paralysis of ciliary activity by bacterial toxic products, edema, etc. It is our object to throw some light on the mechanism of infection and resistance in the upper respiratory mucosa.

PRODUCTION OF INFECTION IN AN APPARENTLY HEALTHY
MUCOSA.

Is previous injury necessary for the establishment of an infection in a ciliated mucous membrane? A virulent hemolytic streptococcus producing small pinpoint colonies and a wide area of hemolysis on blood agar was isolated from a spontaneous epidemic of orbital cellulitis in guinea pigs. The experimental animals were kept in cages in a well heated room to prevent chilling or other effects which might reduce resistance.

A total of twelve young, healthy guinea pigs appearing normal in every respect were inoculated with the virulent hemolytic streptococcus. Two to four inoculations were made on successive days into the nostril, using one to two drops of a twenty-four hour blood agar plate culture, washed off in about five cc. of physiologic saline. The inoculations were made with a round tipped capillary pipette, using great care to prevent any trauma. The pipette was never projected into the nostril beyond the vestibule. The results are shown in table I:

TABLE I.

Pig No.	1	2	3	4	5	6	7	8	9	10	11	12	13	14
Inoculated with.....	S	S	S	S	S	S	S	S	S	S	S	S	SS ³	St. 111
No. of inoculations.....	4	4	3	3	3	3	3	2	2	2	2	2	4	4
Died in days after first injection	5	7	4	4	5	5	6	8	12	9	5	10	lived	lived

S.—Hemolytic streptococcus OC.

S. 83—Hemolytic streptococcus isolated from hypertrophied tonsils.

St. 111—Staphylococcus aureus hemolyticus isolated from chronic ear infection.

There were absolutely no symptoms in animals 13 and 14. All of those inoculated with the virulent OC strain of streptococcus died.

In addition to the above inoculations, seven rabbits were inoculated with the virulent hemolytic streptococcus OC in a similar

manner on two successive days. These rabbits had previously received from four to seven intranasal inoculations of 1 per cent bile over a period of about one month. Previous tests had shown that 1 per cent bile produced in the excised rabbit membrane only temporary slowing of the ciliary activity with complete recovery after washing in physiologic saline. Two of the rabbits died in three days, two in four days, one in six days and one in seventeen days. The remaining animal lived but suffered from a chronic nasal discharge over a period of many weeks.

It is evident from the guinea pig inoculations that those receiving the fewer injections tend to survive longer. This would suggest that the frequency of exposure has, in many instances, a bearing on the course of the disease. The first inoculation may injure the membrane, but at the same time the inoculum be largely or completely destroyed. A second exposure, before there is time for complete recovery of the membrane, would result in the establishment of infection by the same or a different micro-organism. Chronic infections by bacteria of low virulence may frequently have their origin with injury of this type.

The variation in survival time for the rabbits indicates individual variation in resistance. Since nasal infection was evident in all instances within a few days after the first inoculation, only those factors must be involved which constitute nonspecific local resistance following penetration of the membrane, namely, phagocytosis and local reaction. Accidental factors may be involved, such as penetration of a larger quantity of inoculum, which tends to modify the result.

A record of eighteen deaths and one severe infection as a result of dropping, without trauma, a virulent bacterium on an apparently healthy mucous membrane, indicates that a sufficiently virulent strain is able to penetrate the normal mucosa without assistance. Hemolytic streptococci could be demonstrated in heart's blood in a high per cent of cases.

RELATION OF BACTERIAL TOXIC PRODUCTS TO INVASIVENESS.

Bail¹ showed the relation of accompanying toxic products of growth to the virulence and invasiveness of the tubercle bacillus

in the peritoneum of the guinea pig. Pigs receiving tubercle bacilli, plus peritoneal fluid from an infected pig, died earlier and with more extensive involvement than pigs receiving tubercle bacilli alone. This may be a factor of considerable importance in the spread of respiratory disease through droplet infection.

A virulent bacterium like the hemolytic streptococcus OC might reasonably be expected to produce, during its growth on blood agar, toxic substances which would greatly enhance its ability to penetrate the respiratory mucosa. Two groups of ten guinea pigs each were selected which were healthy and free from signs of infection. Hemolytic streptococcus OC was grown on blood agar plates as before and washed off at the end of twenty-four hours in physiologic saline. The saline suspension was divided into two equal parts and one-half washed three times in physiologic saline, or until the supernatant fluid was Biuret negative. One group of pigs was inoculated with the washed suspension of bacteria and the other with the unwashed, care being taken to drop, without trauma, about the same amount of bacteria onto the mucous membrane of the nose of each pig.

Table II gives a summary of the results secured:

TABLE II.

Pig No.	Washed			Unwashed		
	No. Inoculations	Days after first Inoculated for appearance of Symptoms	Days after first Inoculated for appearance of Death	No. Inoculations	Days after first Inoculated for appearance of Symptoms	Days after first Inoculated for appearance of Death
1	4	5	8	3**	1	4
2	4	6	8	3	2	4
3	4	6	8	3	3	5
4	4	3	5	3	3	5
5	4	3	5	3	3	6
6	4	3	7	3	3	8
7	2	3	12	2	3*	12
8	2	5	Survived	2	4*	9
9	2	4	Survived	2	2*	5
10	2	4	Survived	2	2*	10
Average		4.2	7.6		2.6	6.8

*Progressive systemic weakening from fifth day. This was not observed in the corresponding washed group although severe local reaction occurred.

**All 6 distinctly infected after three days and therefore last inoculation not given.

It will be observed that in spite of the added inoculation for six of the animals in the "washed group" and the survival and complete recovery of three animals, there is still an appreciable difference in the average time before the appearance of symptoms and the average time before the death of the animals in the two groups. The figures do not adequately express the difference observed. The manner in which the growth products of the streptococcus aid in establishing infection of the mucosa remains unexplained.

EFFECT OF BACTERIAL GROWTH PRODUCTS ON CILIARY ACTION.

The following experiment was performed in order to determine the effect of the products of bacterial growth on ciliary action, under conditions resembling as nearly as possible those found on the mucous membrane. A 1 per cent Wilson mucin, which is practically free from peptone and other substances digestible by pepsin and acid, was made up in distilled water and sterilized by autoclaving. The reaction was adjusted to pH 7.2 after sterilizing. As a preliminary to determining the effect on cilia, tubes of this medium were inoculated with nine strains of hemolytic streptococci including the OC strain, five strains of hemolytic staphylococci, three strains of the diplococcus of ulcerative colitis and one influenza bacillus. The tubes were incubated forty-eight hours at 37° C. The staphylococcus strains produced an increased cloudiness of the medium, and plating of the sediment from all the tubes after four days, showed all the staphylococci and the three diplococci of ulcerative colitis to be viable in great numbers, while there was no growth from the hemolytic streptococci and the influenza bacillus at this time.

Sørensen titrations of the supernatant fluid from each tube after four days showed no increase in amino groups over the control tubes. This would indicate that any bacterial growth taking place was accomplished by making use of accompanying impurities and not by splitting the mucin molecule. Goldsworthy and Florey² concluded from extensive experiments that any inhibitory properties of mucus are due to its mechanical properties. Waksman and Starkey³ found that proteins combined with nitrogen-free substances are much more resistant to bacterial action. Mucin

consists of a complex nitrogen-free radicle combined with a protein.

Three tubes of the mucin medium were inoculated with *H. influenzae*, *Strep. hemolyticus* (OC) and a selected strain of *Staph. aureus*, respectively, and the tubes incubated forty-eight hours at 37° C. They were then centrifuged at high speed and the supernatant fluid used for testing the effect on ciliary action, using membrane freshly removed from a rabbit sinus. A preliminary direct microscopic observation of ciliary activity was made with the membrane in physiologic saline. The saline was then replaced by the desired fluid without moving the preparation. Table III gives the results:

TABLE III.

Bacterium	Trial	pH of Fluid	Ciliary action stopped
<i>Streptococcus hemolyticus</i> OC.....	1	6.2	46 minutes
<i>Staphylococcus aureus</i> ..	1	5.0	2 minutes
	2	5.0	2 minutes
	3	5.0	1-2 minutes
	4*	7.4	Good more than 1½ hrs
<i>H. Influenza</i>	1		19 minutes
Saline control.....	1	7.4	Good activity more than 4 hrs.

*pH adjusted with NaOH.

Fluid from the same tube was used for each of the four trials with *staphylococcus*. The reaction was adjusted colorimetrically. It appears that acid products of bacterial action on the mucin medium were responsible for the deleterious effect on ciliary action and that the harmful effect was destroyed by neutralization with NaOH. Fluids from the *streptococcus* and *influenza bacillus* were adjusted to pH 7.3 and good ciliary activity observed to continue longer than thirty minutes, which was the period of observation.

Unsuccessful attempts were made to restore the activity of cilia stopped by the *staphylococcus* fluid.

Hydrogen ions have a greater effect on the speed of the ciliary beat than any other ion. Also the H^+ -ion concentration varies greatly in nature, while other ions remain almost constant. Gray⁴ found that acids which penetrate more rapidly into the cell are more effective depressants of ciliary action than acids which penetrate less rapidly, such as H_2CO_3 . Younge⁵ found that the concentration of H^+ -ions necessary to bring the cilia of *Mya* to rest depends on the concentration to which they are normally accustomed. Haywood⁶ showed that acidity produced by HCl becomes more effective in depressing and stopping ciliary action of *Mytilus* as the CO_2 concentration in the fluid increases.

Experiments with excised rabbit membrane showed that a pH of approximately 4.0 produced by bubbling CO_2 through physiologic saline, caused the cilia to stop beating in fifteen to forty minutes. A similar reaction produced with HCl caused stoppage in two to five minutes. A pH of 6.0 or above produced with either HCl or CO_2 allowed activity to continue one to many hours.

Bacterial products may have an effect on the normal ionic equilibrium in a mucous membrane. Gray⁴ observed that ciliated gills of *Mytilus* begin to break up with dissolution of the intercellular matrix and absorption of water by the cells, when deprived of Ca and Mg. Anions are important only as they affect the concentration of cations. Tartrates and citrates, for example, convert Ca and Mg to an unionized condition and thus indirectly affect the membrane.

DEVELOPMENT OF RESISTANCE TO STREPTOCOCCUS INFECTION.

An attempt was made to produce a vaccine immunity in rabbits by applying the vaccine directly to the portal of entry, or the upper respiratory mucosa. It was believed that this method, in accordance with the views of Besredka,⁷ might lead to a higher degree of protection than intravenous immunization.

Since absorption from the respiratory mucosa is very rapid, as shown by Colin,⁸ and high antibody titres in the blood were secured by Pfenninger⁹ and D'Aunay¹⁰ after intratracheal injection of antigens, the injection of a vaccine into the nasal sinus would

seem to give the advantages of both local and systemic immunization. Hopkins and Parker¹¹ gave rabbits intravenous injections of streptococci over a period of eight months without rendering the animals refractory. A systemic immunity to streptococci is, therefore, often very difficult to produce.

A vaccine was prepared as follows: The twenty-four hour growth of streptococcus hemolyticus OC on blood agar was washed off in physiologic saline and, after adjusting to standard density, the organism killed by heating to 56° for thirty to sixty minutes. Injections of 0.5 to 1 cc. were made into the maxillary sinus or sinus and nasal cavity a few minutes after an injection of 1 to 2 cc. of 1 per cent ox bile. The effect of the bile was to help dissolve the mucus layer and to slow down ciliary action, thus giving the vaccine better contact with the membrane and a better chance for absorption. It was found by preliminary experimentation that a 1 per cent bile accomplishes this purpose to best advantage without resulting in permanent damage to the membrane. The following table shows the effect of bile on ciliary action in the rabbit sinus mucosa as observed directly under the microscope:

TABLE IV.

Bile Dilution	Effect	Remarks
1:10	Stopped in 2 min.	Washed in saline, no recovery in 3 hrs.
1:100	Very slow in 20 min.	Washed in saline. Recovered at once
1:1000	Slowed in 3¾ hrs.	
1:10,000	Slow in 4 hrs.	

Inoculations of vaccine were made four to six times over a period of about thirty days. A total of thirteen immunized rabbits and twelve nonimmunized rabbits were inoculated intranasally with about 0.1 cc. of virulent streptococcus OC culture and allowed to live until death or until recovery was established. The following table gives a summary of the results:

TABLE V.

Result	Number of Animals			
	Immune		Non-immune	
	No.	%	No.	%
Surviving without symptoms.....	4	30.7	0	0
Surviving with symptoms.....	1	7.7	1	8.3
Surviving 10 days.....	0	0	1	8.3
Surviving 8 days.....	2	15.4	0	0
Surviving 6 days.....	3	23.1	0	0
Surviving 4 days.....	1	7.7	2	10.7
Surviving 3 days.....	2	15.4	7	58.3
Surviving 2 days.....	0	0	1	8.3

These figures indicate that some of the animals were effectively protected while others were given a certain amount of protection as shown by the increased length of life and symptoms as compared with controls. One of the twelve normal animals survived the inoculations but suffered from a chronic nasal discharge, while three of the thirteen immunized animals died within the usual time limit for normal animals.

Clinical Findings.—A number of interesting clinical observations were made. Of twenty-seven immunized rabbits in all experiments, only four developed a purulent sinusitis. Three of these lived seventeen, thirteen and six days, respectively, after the first inoculation with the hemolytic streptococcus, thus giving plenty of time for the establishment of secondary infection. The fourth animal was killed on the third day following inoculation, and pathologic examination of the sinus membrane showed great masses of eosinophils. Nine of the nineteen nonimmunized rabbits showed a distinct purulent sinusitis in spite of their short average span of life following inoculation. Also twelve guinea pigs which had not been immunized developed pus in the maxillary sinus after being infected.

Septicemia and meningitis were frequent complications. Hemolytic streptococci were found in the hearts' blood in nine of the nineteen nonimmunized rabbits and in eight of the twenty-seven immunized rabbits. Paralysis of the hindquarters usually oc-

curred before death, and microscopic examination of the spinal cord in some of these cases revealed the presence of a meningitis.

Relatively resistant animals showed a tendency to develop local complications, such as erysipelas of the ear, submaxillary cellulitis and pericarditis. There were two cases of erysipelas, two with submaxillary swelling and a number with pericarditis, all among immunized animals. A similar phenomenon was noted by Julianelle and Rhoads¹² working with pneumococci.

Nasal discharge was a symptom common to practically all the animals developing signs of infection. An edematous condition of the mucous membrane, however, was more frequent in the nonimmunized infected animals.

Four of the immunized rabbits lived and developed no symptoms whatever following inoculation.

Microscopic Findings.—One of the most striking differences between the mucous membranes of the nonimmunized and immunized rabbits is the presence in the submucosa of the latter of numerous large masses of mononuclear cells. There are also many



Fig. 1. Showing normal mucous membrane from rabbit sinus ($\times 600$).

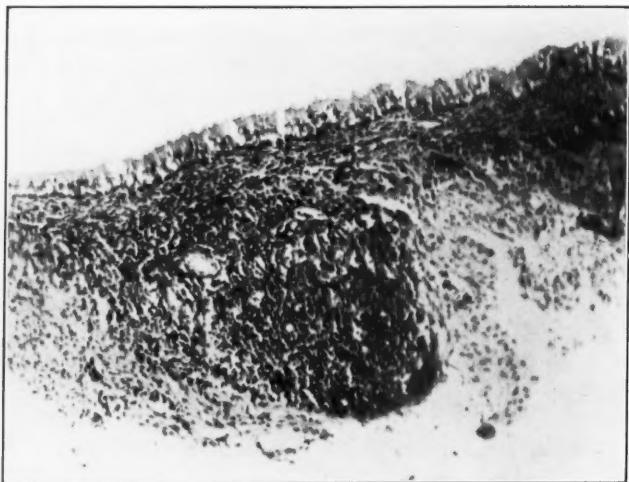


Fig. 2. Sinus mucosa from immunized rabbit before infection. Showing large groups of mononuclear cells and increased mucus production ($\times 400$).

mononuclear cells scattered throughout the tissue. These cells are found in immunized controls before infection with the living hemolytic streptococcus and are equally prominent but more scattered in the tissue after infection. Large groups of mononuclear cells are shown clearly in the accompanying photograph. These are much more numerous than the lymphoid nodules normally found in the sinus mucosa.

Polymorphonuclear neutrophils were always present in few to large numbers within three days after first inoculation with the living streptococcus. They were relatively scarce, however, in some of the more virulent infections. Gay, Clark and Linton¹² found an influx of polymorphonuclear neutrophils to be an early manifestation of infection by streptococci in the pleural cavity of rabbits. The accumulation of groups of mononuclear cells in the mucosa is probably an indication of increased local resistance to the streptococci. The tissue culture work of Parker¹⁴ showing the formation of macrophages from connective tissue cells indicates the probable local origin of these cells.

The sinus mucosa of control rabbits was heavily infiltrated with polymorphonuclear cells in most cases. The lack of polymorphonuclears indicates a virulent infection and a negative chemotactic influence on phagocytic cells.

It is interesting to note that in several of the infected rabbits, both immunized and nonimmunized, large numbers of eosinophils were found in the sinus mucosa when stained by the Giemsa method. The phenomenon cannot be explained on the basis of sensitization instead of immunization, during the period of vaccine inoculations, since it occurred in both immunized and nonimmunized animals.

There are other microscopic changes which may be regarded as defensive measures of a nonspecific nature and which may vary from one individual to another. In this connection may be mentioned (1) a thickening or piling up of the epithelial layer of cells, (2) vacuolation of the epithelial layer, probably with mucus, and (3) a sloughing of the epithelium. In many instances, also, but more particularly in the immunized animals, there was a tendency to hyperplasia of the basilar layer of epithelial cells, indicating an epithelial proliferative stimulus.

Stained sections of the mucosa from many of the infected sinuses showed a mucoid degeneration, especially of the basilar epithelial cells. The columnar epithelial cells in these cases had the appearance of undergoing separation from the basement membrane. Mucoid vacuoles could be distinctly made out in a few instances in the base of columnar cells. The vacuolation appeared to be extending toward the free end of the cell. A phenomenon of this type was observed by Bauer and Chinassi Hakki¹⁵ in the epithelial cells of the gall bladder following ligation of the cystic duct and aspiration of the bile. Under such conditions almost the entire epithelial layer of cells underwent mucoid degeneration in the course of weeks, and a thick layer of highly viscous pseudomucin covered the epithelium. One modification of the mucoid change in the sinus epithelium is observed in Figure 3, where there is thickening of the epithelium with mucoid vacuoles scattered throughout.

Direct microscopic examination of infected sinus membranes immediately after removal revealed instances where the surface



Fig. 3. Showing thickening and vacuolation of epithelium ($\times 600$).

epithelium was thickly covered with fine mucus or mucoid droplets five to seven microns in diameter. The droplets were being hurried along rapidly by ciliary action. The surface tension in such a film and its tendency to bind bacteria would be tremendous. Hilding¹⁶ has shown that mucus is removed by ciliary action more readily than pus or watery substances.

EFFECT OF DILUTION ON VISCOSITY OF MUCIN.

There are some observations brought out in the above work which tend to ascribe to mucus production an even more important defensive rôle than to ciliary action. The normally highly viscid layer of mucus covering the epithelium of the upper respiratory tract offers a very efficient barrier to the entrance of bacteria. Most infections of the mucosa are accompanied by a copious nasal discharge, which, in the early stages at least, is much thinner than normal. An experiment was run to determine the relationship between the dilution of mucin and its viscosity. A 10 per cent solution of Wilson mucin was prepared in distilled water. Dilutions were made from this to give 5, 4, 3, 2, 1, and 0.1 per cent

concentrations respectively. The dilutions were kept in a moist chamber to prevent evaporation until ready for use. The viscosity was determined by means of a Hiss viscosimeter.

The higher concentrations possessed such a high viscosity that it was impossible to make an exact reading on the apparatus. The results in the 4 per cent and 5 per cent concentrations therefore are approximations secured by interpolation.

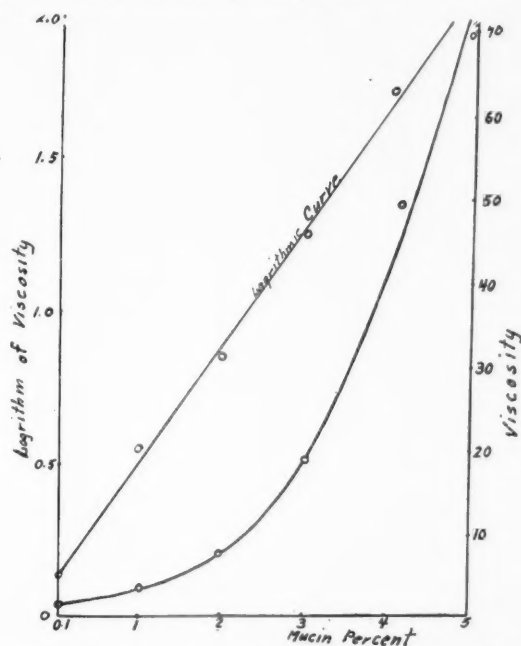


Fig. 4. Graph showing arithmetic and logarithmic viscosity curves for mucin in concentrations from 0.1% to 5%.

An enormous increase in viscosity occurs in the concentration range between 3 per cent and 5 per cent, and a very appreciable increase between 2 per cent and 3 per cent. Preliminary nitrogen determinations by Buhrmester¹⁷ at these laboratories indicate that the mucin content of normal human nasal mucus is between 2 per cent and 3 per cent. This is the range of transition where a slight amount of hydration or dehydration will have the most pro-

nounced effect on viscosity. However, as indicated by the curve, increasing the hydration will have a much lesser effect on viscosity than decreasing the hydration. These observations may have an important bearing on the penetration of an infectious micro-organism into the mucosa. It would be interesting to know whether the secretory cells increase the mucin output when the amount of secretion is increased, thus tending to maintain a constant viscosity. If such a mechanism exists the thin secretions found in the early stages of the common cold indicate that it is soon exhausted.

SUMMARY AND DISCUSSION OF RESULTS.

Evidence is presented to show that a sufficiently virulent bacterium may penetrate the normal respiratory mucosa without the aid of other influences tending to reduce resistance. The animals, consisting of twelve guinea pigs and seven rabbits, were all apparently normal. They were kept under favorable environmental conditions. Eighteen deaths and one chronic nasal infection in a rabbit resulted from the inoculations. Another strain of hemolytic streptococcus and a staphylococcus aureus hemolyticus failed to give any symptoms whatever when inoculated in the same manner into guinea pigs. Penetration of the mucous membrane under such conditions must take place as a result of properties inherent in the bacterium itself.

In considering what these properties may be, one turns naturally to the possible effects of accompanying toxic products. It was found by washing the virulent strain of hemolytic streptococcus free from products of its growth on blood agar, that three out of ten guinea pigs recovered completely from the infection, and the remainder developed symptoms later and lived longer, on the average, than animals in the control group. Infection was not prevented, but the symptoms were delayed and milder than in animals inoculated with bacteria accompanied by their growth products. The presence of toxic growth products is not necessary for penetration of the bacterium into the mucosa, providing the dosage is sufficient. Such penetration is delayed, however, giving the body a chance to bring into action the phagocytic defenses, possibly with greater vigor than usual, due to the decreased nega-

tive chemotactic influence exerted by the bacterial products. Zinsser¹⁸ states that bacteria showing the greatest negative chemotactic influence on phagocytes are the most virulent.

A sufficient amount of toxic substances may remain adherent to the bacteria in spite of washing, to stimulate the mucosa and interfere with ciliary action. Again the bacteria in large numbers may be able to act rapidly enough to produce such an effect before they can be removed from the membrane. The alternative would be for the lashing action of the cilia under normal conditions to cause the bacteria to be mixed with the mucus and a certain number of them lodged on the surface of the cells near the base of the cilia. Here they might remain long enough to cause destruction of cells and growth by extension into the tissues. Careful study with serial sections should throw more light on these questions.

Products of bacterial growth under certain conditions were found to have a deleterious effect on ciliary action. The harmful effect of a staphylococcus fluid was neutralized by bringing the reaction of the fluid to approximately blood reaction. The virulence of the bacterium could not be predicted from the effect of its toxic products on ciliary action. This leads one to believe that some factor is of greater importance than ciliary action to protection against invasion. This factor could readily be mucus secretion.

A certain degree of resistance to infection through the mucosa by a virulent strain of streptococcus was developed by making four to six inoculations with a specific vaccine following inoculation with 1 per cent bile. Large accumulations of mononuclear cells in the submucosa of the sinuses was an important microscopic finding in such immunized animals. An influx of polymorphonuclear cells usually occurs soon after inoculation with the virulent streptococcus.

Agglutinins could not be demonstrated in the blood of immunized rabbits. The indications are, therefore, that the increased resistance developed is largely local.

Three rabbits which were alkalinized by injecting 40 cc. of 5 per cent sodium bicarbonate, died in one, two and two and one-half days, respectively, with the usual symptoms following inocu-

lation with living streptococcus culture. The rabbit, dying within twenty-four hours, showed hemolytic streptococcus in the heart's blood. The resistance of these animals was reduced, allowing rapid penetration of the mucosa and systemic invasion by the streptococcus.

An experiment to determine the effect of dissication or hydration on the viscosity of mucin shows a rapid increase in viscosity as the concentration of mucin increases. The logarithmic curve of the viscosities is a straight line. Calculations from nitrogen determinations indicate a normal mucin content of 2 per cent to 3 per cent in human nasal mucus. On this basis dessication would result in a greater change in viscosity than would be secured by an equivalent amount of hydration.

Any conclusions as to how the normal mucosa is penetrated by a virulent micro-organism would not be justified. It is not even certain that the original penetration is through the ciliated mucosa, although clumps of bacteria were observed in the submucosa beneath the ciliated epithelium. However, certain significant deductions may be made. Bacterial toxic products may have an inhibitory effect on ciliary action and thus create an area of stagnation through which infection can more readily occur. But since penetration, in large doses at least, occurs when the bacteria are washed free of toxic growth products, and since the effect of bacterial filtrates on ciliary action does not parallel virulence, and since the cilia were found to be quite active in the sinuses of many infected rabbits, it is obvious that other factors are of importance. The most important is probably mucus secretion. The exact effect of the streptococcus on mucus secretion has not been determined, but from clinical observations it appears that the amount of secretion and its hydration is increased.

A large quantity of thin secretion would have the advantage of a washing effect and a more rapid removal of a heavy inoculation. The decreased viscosity of the secretion, however, may allow admixture of bacteria in the mucociliary layer and subsequent infection, although such infection resulting from a smaller inoculum may be less severe.

The dragging effect of the mucus layer mentioned by Hilding¹⁹ could not occur to any extent when the mucus is greatly hydrated

as in many acute infections. Growth and multiplication of bacteria at the base of the cilia might then occur with destruction of cells.

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VII.

THE VERTICAL SEMICIRCULAR CANALS IN THE LIGHT OF RECENT WORK.*

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MONTREAL.

Labyrinthine reactions are usually studied by subjecting an animal to different tests which are more or less generally used. Lately, however, we have been impressed with how much information about the condition of the labyrinth can be derived by just watching a frog during spontaneous movement or while it is at rest. The labyrinth is constantly signaling during movement so that any lesion is evidenced by some abnormality of the movement or of the subsequent resting pose.

Recently it has become possible to eliminate any single labyrinthine receptor in the frog by severing its nerve supply and to observe the effect of its absence upon the animal's posture and reactions; it is equally possible to do the converse experiment, viz., to leave intact any one receptor, the remaining portions of the labyrinth being removed, and to observe the effect of stimulation of this lone labyrinthine receptor.

This work is all carried out in conjunction with Professor Tait and it is due to his untiring efforts that our methods of observation and examination have been so much improved.

In this short communication an attempt will be made to demonstrate only one aspect of the matter—the importance of the vertical semicircular canals in controlling the normal tone and posture of the head, especially during movement.

There are three canals in each ear, situated at right angles to each other, and each canal has a dilated portion at one end, in which is situated the nerve end organ—the crista.

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By invitation before the meeting of the American Laryngological, Rhinological and Otolological Society, Atlantic City, May 23, 1932.

The mechanism of stimulation is, that a head movement carries the canal with it and by inertia moves, relatively to the contained endolymph, the hair cells of the crista in the ampullated end of the canal. The result is a nerve message from the appropriate canal to the musculature of the head, body and limbs. A vertical canal appears to be stimulated only by a movement in the direction in which its ampullated end points. A canal responds best to a movement in its own plane, but some of our recent experiments have definitely shown that a canal will also respond to any turning movement even in a plane at right angles to its own. The inference from this last fact is that in the intact animal all six canals may be concerned in any movement and are necessary for its normal completion. The reaction from a normal vertical canal is immediate and so designed as to maintain the head in the horizontal plane.

Ewald (1892) pointed out that the labyrinth exerts a tonic effect upon the body musculature. Subsequent workers, chiefly Magnus (1924), localized this tonic effect to the otolithic part of the labyrinth. This was based upon the observation that the labyrinthine tonic effect was minimal after centrifuging away the otolithic membranes in a guinea pig.

If a frog's head or chin is supported upon the finger tip the neck muscles are felt to be firm and tonic. If the finger is suddenly withdrawn so that the chin is dropped, the arms are immediately adjusted to protect the head from falling. If an attempt is made suddenly to lift the chin or head upwards by putting the finger under the chin, the lift is immediately resisted; if necessary the hind legs will extend before the head will bend upwards. This sudden "chin-drop" and "chin-lift" serve to bring out the normal muscle tone of the head, body and limbs.

After a utricular lesion of one side, in the frog, there is always a permanent alteration of posture and of muscle tone resulting in a forced position. Because there are no forced or fixed postures after a semicircular canal lesion, we were at first inclined to accept Magnus' explanation of the origin of labyrinthine muscle tonus. However, more careful observation of a frog with a single vertical canal lesion reveals the fact that on coming to rest after any

movement the frog is very likely to have a slight downward lean of the head on the corner of the affected canal, e. g., if it is a right anterior vertical canal, the right fore corner of the head is slightly down. This is not a forced position, because the frog can alter it at will. Examination of the frog by the chin-drop test reveals the fact that the head drops downwards and to the right; in other words, the muscles supporting that corner of the head are less tonic. This altered posture we have called "decanalicate posture" in contradistinction to "de-utriculate posture" which is more extreme and more permanent.

If the lesion is of a single canal, it is possible, by means of decanaliculate posture, to diagnose the lesion by simply watching the frog come to rest after any movement. It is therefore most likely that both the canals and the utricles are concerned in the maintenance of normal labyrinthine tonus.

When a frog with one vertical canal absent (i. e., the ampullary nerve has been cut) is placed on the floor or in a tank, it may assume a symmetrical posture, or more likely, if it moves around it will come to rest with that corner of the head dipping down slightly which corresponds to the absent canal. When it is hopping around it will be noted that it may stumble in the direction of the absent canal. When it jumps it tends to land on the damaged corner, so to speak, e. g., if it is a right anterior vertical canal lesion it would land on the right ramus of the lower jaw. From these observations one can say which canal is missing.

If two adjacent vertical canals are eliminated, the animal shows very characteristic reactions during spontaneous movement. If the two anterior vertical canals are absent the frog will sit with its head down. If in another frog the two posterior canals are missing it may sit with its head cocked up. During movement this last frog will throw its head backwards suddenly as though it were stumbling. On attempting to jump sometimes it will turn a somersault backwards. The head seems to be thrown backwards and the arms extended forward. During swimming such a frog tends constantly to be on the surface and jumping out of the water.

If all four vertical canals are removed in an otherwise intact frog it may dip or stumble forwards or backwards or from side

to side during movement and on coming to rest it may sway backwards and forwards before settling down and then its resting pose may be the decanaliculate posture of any one or any adjacent pair of vertical canals. The backward and forward sway of this frog becomes more marked when it is set in shallow water. When swimming in deeper water this frog may sway from side to side, so that it will show the white of its belly alternately on each side. These swaying and oscillatory movements are quite characteristic of this special lesion, absence of all four vertical canals.

A striking experiment is to remove a diagonally opposite pair of vertical canals. The pose may be characteristic of an anterior or of a posterior canal lesion, but it will be noted that one time it is the pose of an anterior canal lesion and a few minutes later it is the pose of the diagonal posterior canal lesion. On attempting any movement there may be stumbling forwards or backwards in the plane of the absent canals, whereas in the opposite diagonal the animal is always steady. On coming to rest the frog may make oscillatory movements in the plane of the missing canals. This effect is best brought out when the frog is in shallow water.

These experiments are striking evidence of the fact that the vertical semicircular canals are constantly being called upon during any movement to maintain the head in the normal steady horizontal position.

Our first impression was that the dipping and swaying which occurs in the absence of a vertical canal was owing to the fact that because the rapid protective response normally set up by the canals was missing, the head dips in the direction of the movement and that only later does the more slowly reacting utricle come to the rescue and initiate the proper protective response. However, it was later shown that if the utricles are removed together with the vertical canals, these dipping and oscillatory movements do not occur. The animal is like a delabyrinthized frog: its movements are irregular but there is no characteristic dipping or oscillation.

To any movement about a horizontal axis, an animal with only two utricles intact immediately sets up the swaying and oscillatory movements previously described. It would seem that in the absence of the vertical canals the head may move out of

the horizontal plane and this allows the utricles to set up to-and-fro reactions which are altogether disturbing to the animal. When the vertical canals are intact the initial movement of the head out of the horizontal plane is prevented and the utricular responses are kept within normal limits. Each of the four vertical canals is a sentinel at its own corner of the head, and acting together with the utricles in control of the head, body and limb musculature, they maintain the normal erect posture of the head during movement or at rest.

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VIII.

HEMATOLOGY AND THE BLOOD DYSCRASIAS IN RELATION TO DISEASES OF THE EYE, EAR, NOSE AND THROAT.*

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PHILADELPHIA.

Hematology and the blood dyscrasias fully deserve a place, perhaps even the first place, in a symposium, for progress in this field has been noteworthy, and hematology has become a feature of the proper study of each and every patient.

The normal blood picture is maintained by a perfect balance between production and consumption of each cell type. Production proceeds under the control of stimuli, about which we know little, and the various blood cells grow to an appropriate age in the shelter of their respective hemopoietic homes before entering the busy world of circulating blood. For this maturation a whole series of stimuli, some hormonal perhaps, seem to be necessary.

Proper maturation permits only sufficiently adult cells to appear in the blood and, if production and consumption are balanced, there results what we accept as the normal blood picture. We have only recently, however, recognized that basal conditions are just as necessary for a constant blood count as for a metabolism determination. Wide variations in the white and red cell counts occur under the activities of daily life; the basal white count, for example, may double from muscular activity. This must be kept in mind in practice.

The other factor, destruction or consumption of the various cell types, proceeds in different ways: erythrocytes are phagocytized by the reticulo-endothelium not only in the liver and spleen but wherever this widely distributed tissue occurs; while the senile white cell is now believed to pass into the digestive tract

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chiefly through the mucous membranes of the mouth. This perhaps suggests that the constant involvement of the gums, mouth and pharynx in leucemia is analogous to the splenic enlargement of the anemias with increased blood destruction.

Little is known of the fate of the platelets; they probably are dissolved in the periphery of the body where their potent juices remain to supplement the circulating platelets in time of need.

The abnormal blood picture results from any disturbance of the balance—the so-called hemolytopoietic balance—between production and consumption, or from an added element of abnormality, often termed “degeneration.”

Production may be decreased or speeded up, and maturation may be interrupted. Such an arrest of maturation best explains the pathology and blood pictures of Addisonian anemia and an analogous arrest may be found to explain other important disorders of the blood, for example, the leucopenia which may lead to agranulocytic angina. Progress will come as we learn the successive stimuli required for normal maturation of the erythrocyte, the platelet and the various leucocytes.

The second topic for discussion concerns the common origin of many of the symptoms seen in the group of blood dyscrasias.

No doubt each of the blood dyscrasias is an entity, but certain basic changes in the blood recur frequently in their various blood pictures. Anemia, leucopenia and thrombopenia, for example, are present as primary or secondary changes in many of the dyscrasias, and whenever present are likely to lead to the same symptoms. For example, a low platelet count explains the retinal hemorrhages of such widely different dyscrasias as acute leucemia, aplastic anemia and purpura hemorrhagica. Also tinnitus and pallor may occur in any dyscrasia in which anemia is part of the blood picture.

It is most important for you to realize the disease nonspecificity of such symptoms and to relate them to the basic change in the blood picture rather than to any one clinical syndrome.

Also certain tissue changes may occur in more than one dyscrasia and may cause identical results. For example, tissue

infiltration with abnormal white cells is common to the various leucemias and may, by pressure, cause deafness or other local symptoms. Any leucopenia opens the way to infection.

Of course, local differences in anatomy and physiology influence very much the distribution of such manifestations. Such local determinants are not the same in the ear as in the mouth, nor the same in the eye as in the tonsil. The mouth and the colon as frontiers constantly exposed to bacterial invasions have more in common than the mouth has with its neighbors, the eye and the ear. One is, therefore, not at a loss to understand why the same necrotic lesions often occur in mouth and in colon in agranulocytic leucopenia and seldom if ever in eye or ear.

Futhermore, many of the manifestations of the blood dyscrasias are determined by local circulatory conditions. For example, the peculiar vascularity and visibility of the retina permit the recognition of capillary hemorrhage and of the pathognomonic pictures of polycythemia, intense anemia and leucemia. In contrast, the structure of the ear renders its vessels and nerves very liable to pressure from hemorrhage or infiltration, both of which occur in Addisonian anemia, purpura hemorrhagica and, in leucemia, especially into the labyrinth.

Let us next comment on certain of the dyscrasias—first the anemias. Only a few years ago an authoritative text on medical ophthalmology discussed at great length the eye symptoms of chlorosis, but gave little or no attention to other forms of anemia. Today we seldom see chlorosis and we realize that the ocular manifestations then described for chlorosis are those of any anemia.

Chlorosis was probably due to a faulty dietary, and more and more we appreciate the part played by disturbance of the digestive tract in the production of the anemias. In the Addisonian form the lack of a specific intrinsic factor in the gastric juice, independent of hydrochloric acid or pepsin, leads to a deficiency in a substance necessary for the normal maturation of erythrocytes. Similarly most if not all of the anemias resembling the Addisonian type have a distinct relation to the digestive system, for example,

the anemias of sprue, fish tapeworm infestation, cancer of the ascending colon and certain intestinal strictures.

The interest which formerly centered on the spleen as the mysterious cause of anemia has been transferred to the gastrointestinal tract.

Of course, in any discussion of anemia it is the treatment of Addisonian anemia which overshadows all other progress; no longer can this disease be called either primary or pernicious. There is no more fascinating story of progress than that of the steady march forward from the use of raw liver to today's highly potent extract for parenteral injection.

Early diagnosis and proper treatment will prevent in the future the fully developed picture of Addisonian anemia. No longer will we see the characteristic atrophy of the tongue or the horrible tragedy of posterolateral sclerosis which even liver will not cure. If this complication ceases to occur, I am afraid that most clinicians who still keep tuning forks only to test for the characteristic loss of vibratory sense will put them away in the cabinet with the discarded monaural stethoscope and the cups.

Concerning other anemias there is little need be said. Sickling of the red cells, that peculiar change of shape which occurs in wet preparations, has been found to be more common than was at first suspected and often to occur without anemia. It is clearly hereditary and almost limited to the negro race. It is important for this reason to distinguish another familial syndrome with oval erythrocytes, which occurs in others than negroes and without anemia.

Perhaps you have encountered the latest fashion in classification of anemia: that based on the size of the red cell and its hemoglobin content. Anemias are normo, macro or microcytic, and if the hemoglobin saturation is low, hypochromic, indices based on cell volume are to replace our old friend, the color index. This classification leads to diagnoses such as "chronic hypochromic anemia with achlorhydria and microcytosis." This syndrome resembles chlorosis but occurs in middle aged women and may be analogous to the leucopenia of agranulocytic angina, which is most common in women of that age, and to a form of thrombopenia described by Nagy in women with ovarian dysfunction.

There is nothing to recount of the polycythémias; the frequency of visual symptoms and the characteristic retinal picture have been brought home to us by emphasis in the literature.

The leucemias have been the subject of little advance in knowledge. We are still totally ignorant of the cause of any of the various types, but we do perhaps realize a little better that they are very different diseases, with nothing in common other than the white cell increase. The renewal of therapy with arsenic has not led to any better results than were obtained many years ago.

There has been much recent interest in leucemoid blood pictures, such as occurs in infectious mononucleosis, in the pseudo-leucemic anemia of infants, and in certain unusual reactions to infection best seen perhaps in whooping cough.

The similarity to leucemia rests either on a marked increase in the total white count, on the presence of many immature cells, or on some other change from the normal picture. In contrast to true leucemia, the leucemoid blood pictures can usually be related to a definite cause and are only temporary. The term "leucemoid" is also employed of another group of conditions which find their resemblance in tissue pathology without change in the blood picture. Chloroma, familiar to the ophthalmologist from the intra-orbital tumor deposits, is an example.

Leucopenia is probably the disorder of the blood which at this time is foremost in the minds of many of you, because of its relation to agranulocytic angina.

It has long been known that leucopenia from a reduced number of neutrophils occurs in some infections, including overwhelming infection, in aplastic anemia, in benzol poisoning, sometimes after arsphenamin, and during anaphylactic shock. Hematologists were slow, however, in recognizing a primary, apparently causeless, form of leucopenia analogous to primary anemia and to primary purpura hemorrhagica. It is now realized, however, that in mild degree this is fairly common and may be associated with no symptoms beyond perhaps a tendency to fatigue. In some individuals the leucopenia is chronic; in others, it comes and goes for years, sometimes in quite regular cycles. Only when the leucopenia is

severe do the amazing nonreactive necrotic lesions appear in the mouth, pharynx, bowel, vagina or skin. At first the cart was placed before the horse, but now we know that the leucopenia is primary and the infection secondary. We do not know, however, the cause of the leucopenia; there is no evidence to relate it to the inhalation of exhaust fumes, to chronic poisoning from cans or other food containers, to endocrine dysfunction, all of which have been suggested. Several of my patients with agranulocytic angina were allergic, and this may play a part in bringing about the leucopenia just as allergy probably explains the relative lymphocytosis occurring from neutrophil decrease in sympathetic ophthalmia.

Whatever the cause, we have not yet at our disposal any satisfactory treatment of primary leucopenia. There is some evidence that an arrest of maturation is the pathogenesis, but we know neither the lacking stimulus nor how to supply it. Transfusion has been the therapy of choice, but today nucleotide injections are being widely employed, apparently with some success. Unfortunately, the response is not uniform nor prompt enough to save the fulminant type of case, which is that giving the greatest mortality.

Next a word about the purpuras—not to describe progress—but to admit ignorance. Purpura appears in so many diseases that the phenomenon is no more specific than is anemia. Again we find primary and secondary forms. Rheumatic purpura is merely a symptom of the infection, and a coincident iritis is not to be blamed on the purpura but on a common background, as was emphasized by Benedict¹ in 1930. Any focus of infection may cause both purpura and arthritis, as in the patient reported by Borries,² with antral empyema.

Even when we take into account quantitative and qualitative disturbances of platelets, and deficiencies in calcium and fibrinogen, we are unable to offer a satisfactory classification of the hemorrhagic disorders. That the platelets do not explain the whole matter has recently been strongly stressed by Morawitz.³

Normally there are sufficient platelets to plug any tears in the vascular endothelium and to supply the material essential for coagulation. When thrombocytopenia is present a purpura is

readily attributed to the lack of platelets, but so often purpura occurs in the presence of a normal platelet count as to leave the whole subject in confusion. There is great need of further progress in this field.

Hematology does not find its field of greatest practical value in the identification of rare blood dyscrasias but rather in the study of disorders encountered daily by the physician, the surgeon and the specialist. Sometimes the change in the blood picture is spectacular, as for example, the stippling of erythrocytes in lead poisoning, the lymphocytosis of whooping cough, the eosinophilia of trichiniasis and the monocyte increase of subacute bacterial endocarditis.

Far less spectacular but of much greater importance are the changes which accompany infection. Interest has progressed from the increase in the total white count and the neutrophil percentage to the appearance of immature cells. This latter sometimes gives the so-called leucemoid blood picture, but more significantly it gives an opportunity to gain insight into the presence, progress and prognosis of an infection from the relative ages of the neutrophils present.

Arneth, in 1904, using the nuclear configuration as a basis, tabulated the neutrophils in columns according to age. His method, as well as Schilling's even more complex "hemogram," have been almost abandoned for routine use—but we still speak of a "shift to the left," indicative of an increase in young forms, and a "shift to the right," of old.

Simpler and more practical methods merely divide the neutrophils into two age groups, according to whether the nucleus has or has not reached the stage when the chromatin segments are connected by mere filaments. Normally the cells with nonfilament nuclei average only 8 per cent of the neutrophils and the upper limit in normal blood is 16 per cent. An increase above these figures is a "shift to the left," and indicates an increase in immature forms. This may occur in the absence of any increase in total count and even without any distinct increase in the neutrophil percentage. Under such conditions the method may give very useful information as to the presence of infection, but only repeated observations permit one to draw many conclusions.

A word of warning is necessary; even the simplest of these methods requires care and skill. We must be on our guard not to allow dogmatic reports from the laboratory to lead us to neglect other clinical evidence and our own practical judgment.

Finally, we must mention the usefulness in infection of the determination of the sedimentation rate of the erythrocyte.

Many years ago, during the period when phlebotomy was so copiously performed, one of the main indications for bleeding was an increase in the buffy coat or, as it was called, the "size" of the blood, which depends in a large measure on the rapidity with which the erythrocytes settle before coagulation occurs.

After bleeding became unfashionable, there was no further interest in this until recently. An increased rapidity of sedimentation has been found to have a peculiar value under certain conditions of infection. The test is in no way specific; an increased rapidity is present whenever there is tissue destruction, as in all infections, in fever, with tumor cachexia and in pregnancy.

In spite of this, the test has proved of great value in determining the continuance of infectious activity, especially after fever, leucocytosis and local signs subside, as for example, in pelvic inflammatory disease, pulmonary tuberculosis and rheumatic infection. It seems likely that there are similar uses for this method in your specialties.

There is much more that should be said about hematology, but time allows only a closing paragraph.

Hematology owes its progress to research and to elaborate methods, but in practice the methods of hematology must be such as we can perform, and the information derived must be such as we can understand and apply. You and I, in our daily work, must know the tools we use and what we can count upon them to do for us. We must not be easily led to abandon the tried and true for something merely new, but we must be ready to hear of the new and adopt it if its value is proved.

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IX.

CARDIOVASCULAR DISEASES IN RELATION TO THE RETINA.*

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Liebreich, in 1859, gave the first accurate description and graphic illustration of "albuminuric retinitis." Twelve years later, or sixty years ago, Allbutt, who was primarily an internist, published a book on the use of the ophthalmoscope in certain diseases, including those of the kidney. Allbutt discussed the possible relationship of the retinal changes to renal pathologic processes. He admitted the difficulties in explaining such a relationship, but definitely stated that in his opinion the retinitis occurred late in the course of the renal disease and was of serious prognostic import. Michel, in 1884, was the first authority to state how long a patient might be expected to live after the development of albuminuric retinitis. His own statement was: "In regard to the maintenance of life, the retinitis is of very serious significance; in the majority of cases in which simultaneously retinitis appears and the diagnosis of granular kidney is made, after a course of one-half to one and one-half years death can be expected."

Physicians are indebted to Marcus Gunn, in 1898, and to Foster Moore, in 1916, for distinguishing the arteriosclerotic changes in the retina from those associated with primary renal disease. They were both of the opinion that certain retinal arterial changes were localized lesions of a general arteriosclerotic process occurring throughout the body. Moore showed that there was a relationship between the increase in systolic blood pressure and the extent of the retinal arteriosclerotic changes. O'Hare and Walker, in 1924, pointed out clearly that hypertension exists, or has existed, in any case in which there are sclerotic changes in the

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Work done in the Division of Medicine and the Section on Ophthalmology, The Mayo Clinic.

retinal arterioles. From this they assumed that the condition of the retinal arterioles is an index of the state of the arterioles of the tissues generally.

In October, 1920, I became very much interested in the subject of albuminuric retinitis. A young man was admitted to the hospital with a diagnosis of chronic glomerulonephritis. Both Dr. Wagener and I were convinced that the patient had typical albuminuric retinitis, but much to our surprise his renal function was extremely good, much too good for that seen in cases of terminal chronic diffuse nephritis. This patient was our first recognized case of so-called malignant hypertension. By further clinical, ophthalmologic and pathologic studies, we were able to identify a syndrome, which was characterized by hypertension and diffuse arteriolar changes throughout the body. The prognosis was serious, and depended in large part on the type and extent of the retinal changes. By this work we were led to distinguish, from general and retinal arteriosclerosis with essential hypertension, a special group of cases of late and rapidly progressive diffuse arteriolar disease.

The pathologic changes in the arterioles that have been mentioned usually have been considered organic in nature. The significance of vasospasm without preceding organic changes has recently been shown by Haselhorst and Mylius in eclampsia. They actually demonstrated in a case of eclampsia, by retinal photographs, the presence of arteriolar spasm and the rapid disappearance of the spasm following delivery. In the present year, Koenigsberger and Bannick reported a case, in a male subject, of acute vasospastic hypertensive disease, in which later, so-called malignant hypertension developed. In the early stages in this case accurate retinal studies were not made. Subsequently, Wagener, Burke and Barker described a small series of similar cases in which vasospasm, including that of the retinal arterioles, was a prominent feature. The arterioles of the skeletal muscles in these cases were shown to have intimal proliferation. Therefore, these authors expressed the belief that both vasospasm and actual pathologic changes involved the arterioles throughout the body.

For the understanding of the relationship of changes in the retina to cardiovascular disease it is necessary to consider the

association of hypertension with arteriolar spasm, glomerulonephritis and diffuse arterial disease.

ESSENTIAL HYPERTENSION ASSOCIATED WITH ARTERIOLAR SPASM.

Several observers, including Elschmig and Wagemann, have recorded transient blindness associated with occlusive spastic constriction of the retinal arterioles. With the quick return of vision the spasticity of the retinal arterioles disappeared. A similar case has been followed by Dr. Wagener. Numerical data concerning this and other cases referred to in the paper will appear, for the most part, in the table. The history and general data will be given in the text.

Case 1.—A physician's wife, aged 43 years, gave the history that several years before she had had two miscarriages, with which there had been associated albuminuria. Also for four or five years she had had attacks of petit mal, for two years, white or dead fingers, and for ten years, short periods of partial or complete blindness. At our initial examination, we thought the visual disturbance was probably secondary to general spasm of the cerebral vessels, rather than of the retinal vessels, for moderate essential hypertension was present, accompanied by slight sclerosis, but by no visible spasm of the retinal arterioles. The attacks of petit mal and white fingers were also considered to be due to arterial spasm. While the woman was being examined again, two years later, Dr. Wagener observed with the ophthalmoscope, actual occlusive spasm of the left inferior temporal artery as an attack of blindness occurred. The spasm and blindness persisted for one or two minutes and then the previous conditions were restored. This observation indicated that the temporary blindness was due to retinal vascular spasm and not to cerebral spasm, as we had previously incorrectly thought. Four years later, thrombosis of a retinal vein (superior temporal) developed.

The probable sequence of events in this case was the development of eclampsia with a residual tendency to hypertension and recurrent arterial or arteriolar spasm. The significant fact which should be stressed is that arterial spasm can occur in the retina and tissues generally for a short period and leave no demonstrable injury of tissue. If, however, the spasm persists, an anemic infarct results with permanent loss of vision.

In eclampsia, vasospasm, although not completely occlusive, is an important factor. If it persists long enough, ischemia or venous stasis with secondary organic lesions of the retina develop. Many authorities presume that vasospasm is the initial basis for the renal pathologic changes in this condition. Yet, if

the eclamptic condition is relieved by delivery, the vasospasm may cease and complete restitution of the retina and kidneys may take place; on the other hand, even after delivery, vasoconstriction may persist and the retinal and renal lesions progress. In the case of the physician's wife, just reported, presumably there was a residual tendency to periodic vasospasm. In those cases in which vasospasm persists the primary abnormal physiologic process probably gradually gives rise to pathologic changes in the arterioles. That such a sequence of events may occur during, and subsequent to, eclampsia seems logical, judging from facts known at present. A similar sequence of vasospasm followed by pathologic changes would at least offer a good explanation for the facts observed in the series of cases reported by Koenigsberger

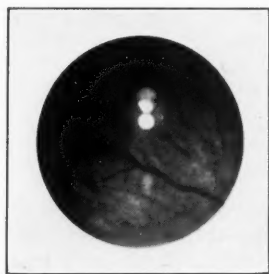


Fig. 1 (Case 2). Thrombosed retinal arteriole (below and to the right of the reflected light from the camera carbons).

and Bannick, and Wagener, Burke and Barker, in which the patients were males and nonpregnant females. Next is an example of such a case.

Case 2.—A man, aged 35 years, gave much direct and indirect evidence of diffuse hypertensive arterial disease. He had had hematuria for two and a half years, hypertension for one year, and headaches and visual disturbances for one year. The retinal arterioles were small and evidently constricted. Arteriolar thrombosis was demonstrable in the retina (Fig. 1). On histologic examination of the arteries and arterioles of muscle tissue obtained for biopsy, there was definite narrowing of the lumen, pathologic changes in the wall, and, in addition, actual arteriolar thrombosis (Fig. 2). Thus, it was possible to demonstrate by the ophthalmoscope and by histologic technic that arteriolar spasm coexisted with actual anatomic changes, and that arteriolar thrombosis had occurred. The right renal pelvis was found to be dilated and moderate secondary anemia was present. The patient died within a few months.



Fig. 2 (Case 2). Arteriole with thickened wall and organized thrombus in pectoralis major muscle.

This case demonstrates how serious the prognosis may be. On the other hand, possible relaxation of the arteriolar spasm may explain the fact that in certain cases of well developed vascular retinitis remissions do occur.

GLOMERULONEPHRITIS.

It is now generally recognized that retinal changes occur only in a small percentage of cases of definite diffuse glomerulonephritis. This fact has become more apparent in the last few years, with the recognition that diffuse vascular disease rather than renal disease is the cause of many cases of so-called albuminuric retinitis. Retinitis is extremely rare in acute glomerulonephritis, and the few instances studied indicate that the findings are similar to

those present in eclampsia. In subacute glomerulonephritis, general edema is often a troublesome symptom. It is in such cases that retinitis characterized by edema of the disc, large "snow-bank" exudates and even detachment of the retina occur. Also, this type of retinitis is seen during an acute exacerbation in the course of chronic glomerulonephritis. In this type of retinitis (Fig. 3), edema is a prominent feature and apparently is a local manifestation of the general disturbance in metabolism of water and salt.

Case 3.—A woman, aged 27 years, gave the history that when she was 23 years of age she had albuminuria and edema, and infected tonsils were removed. Three weeks before I saw her she had a respiratory infection followed by edema. Two days after her admission, she had acute pericarditis (Fig. 3).

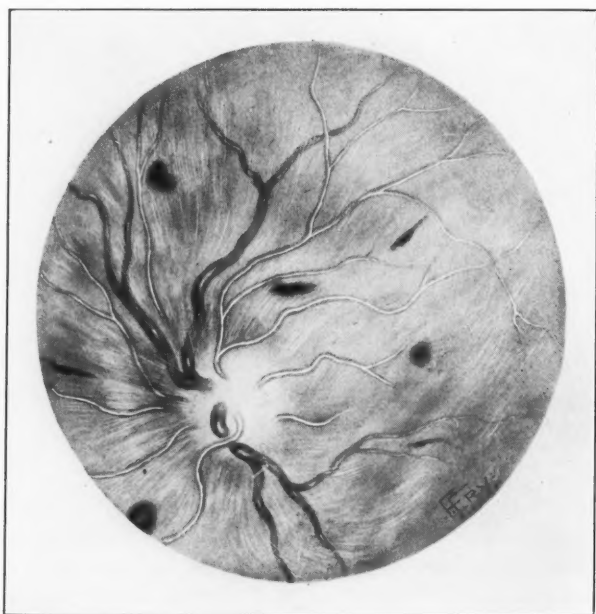


Fig. 3 (Case 3). Draughtsman's representation of diffuse edema and retinitis, in a case of chronic glomerulonephritis, during an acute exacerbation.

Two retinal lesions occur late in the course of chronic glomerulonephritis: angiospastic (albuminuric) retinitis and that associated with chronic anemia. The former is very similar to the retinitis of diffuse arterial disease, but changes in the vessels are less marked.

Case 4.—A man, aged 19 years, had had edema for two months before I saw him, and at that time mild secondary anemia was present. About a month after I examined him the edema had disappeared, and tonsillectomy was performed. But the edema returned and his condition became worse. On the last two days of his life, approximately eleven months after I first saw him, and thirteen months after the beginning of his illness, he was markedly edematous and anemic, and convulsions occurred. Angiospastic retinitis was present in the last six weeks. Necropsy revealed chronic glomerulonephritis.

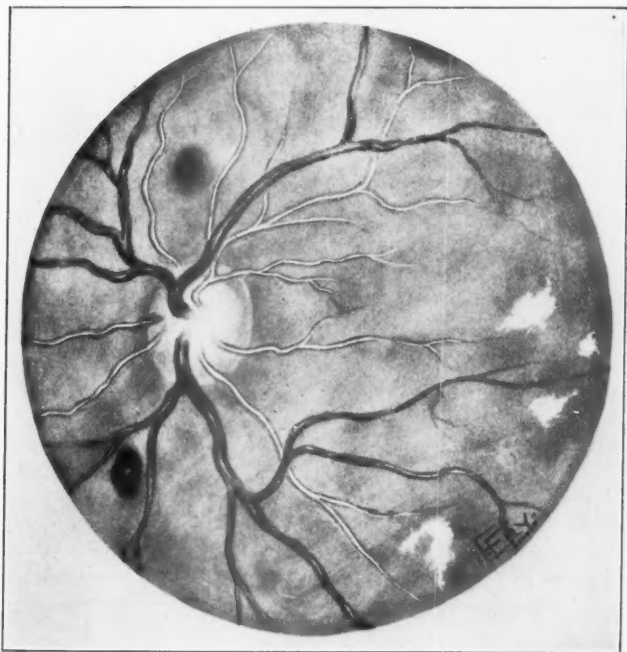


Fig. 4 (Case 5). Draughtsman's representation of retinitis of chronic anemia and sclerotic changes in arterioles. Chronic uremia is also present.

The present conception as to the cause of "albuminuric retinitis" is that it is secondary to arteriolar changes, but that these are less marked than in chronic diffuse arterial disease. They are probably primarily angiospastic rather than angiosclerotic. The presence of this type of retinitis is of very serious prognostic significance.

In cases of chronic anemia due to different etiologic factors, characteristic retinitis can occur. Such retinitis may be present in the terminal phase of chronic glomerulonephritis. If, in addition to this retinitis, there are changes in the arterioles, renal or diffuse vascular disease should be suspected, and if possible excluded. In case 5 (Fig. 4) there was such a type of retinitis.

Case 5.—A woman, aged 59 years, was first seen in 1922. In 1929 she had an adenoma of the thyroid gland, with basal metabolic rate of -6 , pains in the lower part of the legs, and dyspnea on exertion. In 1932, secondary anemia was present, and she had had attacks of loss of consciousness for one year. There was 7.4 gm. of hemoglobin in each 100 c.c. of blood and erythrocytes numbered 3,480,000 in each cubic millimeter of blood. She died later in that year. Necropsy revealed hypertrophy of the heart. The kidneys, of which there was arteriosclerotic atrophy, had a combined weight of 75 gm. Acute pericarditis, also, was present.

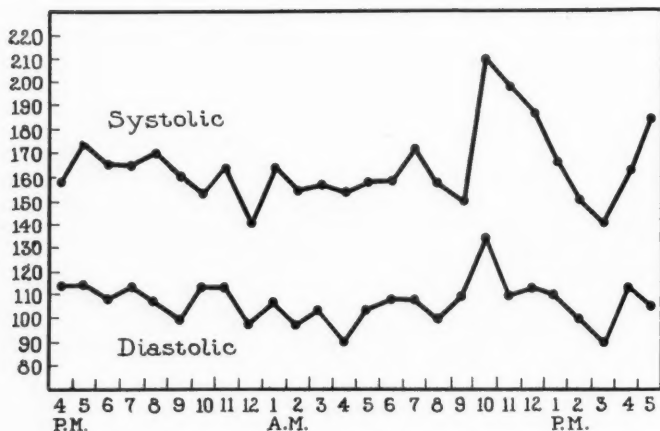


Fig. 5 (Case 6). Blood pressure for twenty-four hours in a case of diffuse arterial disease with hypertension, group 1.

ESSENTIAL HYPERTENSION ASSOCIATED WITH DIFFUSE ARTERIAL DISEASE.

Allbutt, in 1896, clearly described the clinical syndrome of essential hypertension and termed it "hyperpiesia." He recognized that such a condition is compatible with continued good health; indeed, he had observed one of his patients for twenty years. Luckily, at the present time this group of cases constitutes the great majority of those of hypertension, and I have termed it "diffuse arterial disease with hypertension, group 1."* A study of blood pressure for twenty-four hours in one of the cases seen at the clinic (case 6, Fig. 5) demonstrated the decided effect of rest on the hypertension. The retinal changes in this condition are minimal and consist of slight but distinct organic narrowing of the arterioles (Fig. 6).

Case 6.—The patient was a man, aged 38 years, when first seen in 1918. At that time he complained of having had lumbago for five weeks; he also had cardiac arrhythmia. In 1924 the cardiac arrhythmia was still present. He did not have hypertension either in 1918 or in 1924. In 1930 the man was 50 years of age, at which time he had hypertension and headaches, and had noticed increased irritability for two years. He had had an attack of vertigo one month before his return for study. One year, and also two years later, he felt well.

Patients who have a continuously higher blood pressure and more distinct anatomic changes in the arterioles than those of the previous group, but who still are in good general health, have been placed in a second group, called "diffuse arterial disease, with hypertension, group 2."

Case 7.—In 1928, a woman, aged 23 years, was known to have had hypertension and headaches for six months. Arterioles of the muscles were found on histologic examination, to be definitely narrowed. Although she has diffuse arterial disease, her health has remained good for four years.

One's impression of these patients of group 2 is that the disease is of a more serious nature, either due to a more rapid course since the onset, or because they have a later stage of the condition described under group 1. Retinal changes in this type of

*The grouping of these cases into four groups has been an aid in estimating prognosis. Each group has distinct features which may or may not prove to belong to a single disease process. Further study, I hope, will confirm the usefulness of this grouping.

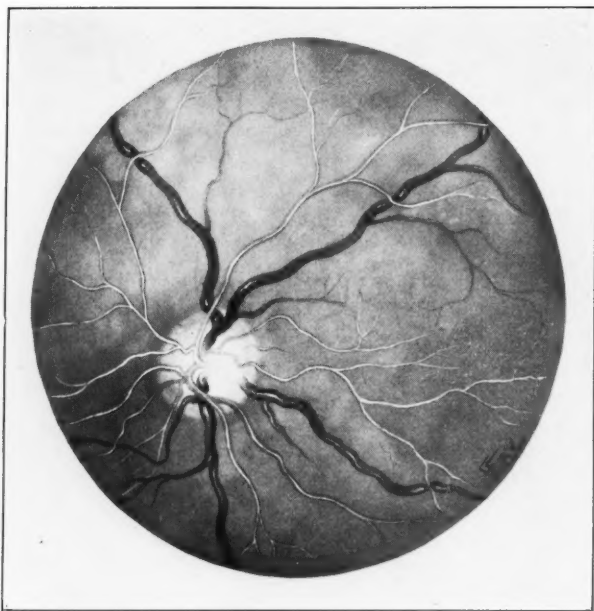


Fig. 6. Draughtsman's representation of early changes in retinal arterioles in a case of diffuse arterial disease with hypertension, group 1.

case are represented in Fig. 7. Follow-up studies in this entire group show that the prognosis is more serious than in group 1.

Group 3 includes cases in which there is hypertension and readily demonstrable diffuse arterial changes, but in which, in addition, there is mild retinitis. Case 8 (Fig. 8) is a good example of the progressive type of diffuse arterial disease. As a group the prognosis in these cases is serious.

Case 8.—A woman, in 1929 was aged 35 years, and at that time was known to have had hypertension for six months. Three years later, headache and pain in the legs had developed. Within these three years the condition had changed from that of group 2 to that of group 3. Studies of blood pressure, as well as retinal changes, indicated that the course of the disease was rapid and progressive.

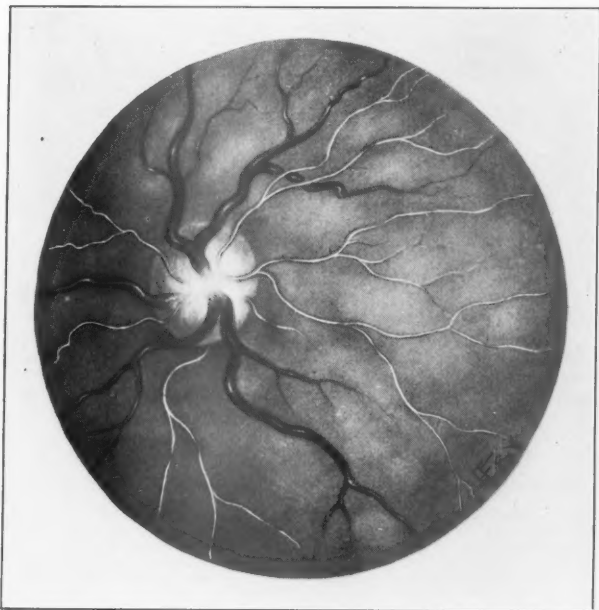


Fig. 7. Draughtsman's representation of changes in retinal arterioles more marked than in Fig. 6. The condition was diffuse arterial disease with hypertension, group 2.

It is of great interest that even in cases of group 3 there may be a remission in the activity of the pathologic process, as in case 9 (Fig. 9).

Case 9.—A woman, aged 31 years, was seen by me for the first time December 12, 1931. She had edema of the eyelids, and knew that she had had hypertension nine years before. Three years before I saw her she had a nasal hemorrhage, and one year previous to this, right hemiplegia appeared. On ophthalmoscopic examination there was evidence of a previously active process and many small arterioles were seen to have become obliterated. Some arterioles of the retina had undergone thrombosis, and on biopsy the same process was seen to have taken place in some arterioles of a skeletal muscle.

Could cessation of arteriolar spasm be responsible for the remission in this case 9?

The use of the term "malignant hypertension" has been adversely criticised by many physicians. To avoid any controversy



Fig. 8 (Case 8). Changes as revealed by the camera in arterioles and retinitis, without edema of disk, in a case of diffuse arterial disease with hypertension, group 3.



Fig. 9 (Case 9). Marked vascular changes as reproduced on photographic film, including thrombosis of arterioles and residual signs of retinitis, in a case of diffuse arterial disease with hypertension, group 3, which was in remission.

over terminology, I have grouped such cases under the title of "diffuse arterial disease with hypertension, group 4." These undoubtedly are the most serious examples of this disease. In the first description of these cases the prognosis was said to be very serious, and the duration of life to be only a matter of months or a year or two. Subsequent studies of a much larger series of cases has confirmed this impression, and led me to the conclusion that the similarity of Michel's statistics in 1884 to mine indicates that the majority of his cases of "albuminuric retinitis" belonged to this group 4. The characteristic findings are diffuseness of arterial and arteriolar thickening throughout the body and the retinal changes. Wagener has emphasized the marked spastic and organic narrowing of the arterioles and the edema of the discs. The presence of the latter he considers the feature that distinguishes this type of retinitis from the retinitis seen in cases of group 3. Case 10 is an example of a rapidly progressive condition.

Case 10.—A man, aged 37 years, when first examined in March, 1932, was up and about. He had had hypertension for two years and headache for eighteen months. Gross hematuria, which had appeared ten days before, caused him to come to the clinic. Slight failure in vision had been noticeable for one year. The appearance of the ocular fundus was typical of cases of group four and diffuse arterial disease was evident



Fig. 10 (Case 10). Retinitis with edema of the disk, as revealed by the camera, in a case of diffuse arterial disease with hypertension, group 4.

(Fig. 10). One month later diffuse pain in the muscles appeared, and then came blurring of vision of the left eye. About two months after I first saw the patient he died from cerebral, cardiac and renal insufficiency. Necropsy revealed hypertrophy of the heart, ischemic nephritis and acute pericarditis.

Not all cases of group 4 are of such rapid course. Some patients even have periods suggesting remission. Such a case, belonging to group 3, was considered previously. Here again spasm of the arterioles may determine whether the condition is progressive. I have observed a few cases of group 4 without histologic changes in the arterioles of the skeletal muscles. Evidence is also accumulating that even when there is marked pathologic change in the arterioles compensatory mechanisms may still permit periods of improved tissue metabolism.

COMMENT.

Since Helmholtz devised the ophthalmoscope, eighty years ago, knowledge regarding vascular changes in the retina has steadily increased. Studies of the ocular fundus have played a significant part in the gradual definition of diffuse arterial disease and in its separation from primary diffuse glomerulonephritis. The actual observation of the normal and abnormal retinal arterioles during life and the knowledge that arterioles in the tissues everywhere may be reacting similarly have given new facts and stimulated new ideas as to the physiologic function of and pathologic changes in arterioles. Abnormal vasospasm can be observed in the retinal

arterioles; when of very short duration no gross changes occur in the retina; if prolonged, secondary retinal lesions develop, and there is evidence that prolonged vasospasm may lead to histologic changes in arterioles. It has been possible to show that vasospasm and definite histologic changes can co-exist. In such a case, as one might expect, there is a tendency for thrombosis to occur in the arterioles. Relaxation or intensification of the arteriolar spasm offers a possible explanation for the remissions and exacerbations that occur in the course of diffuse arterial disease.

The type of retinal vascular changes, and the presence or absence of retinitis, are important aids in arriving at prognosis. At present the conception of the end stages of vascular retinitis is much clearer than formerly. Nevertheless, when serious lesions of this type are observed in the retina, Liebreich's experience in 1859 should be recalled. He observed complete subsidence of a marked "albuminuric retinitis."

There are still many factors in the course of diffuse arterial disease that are difficult to explain. The etiology is unknown. Cases in which the course is rapid and progressive are in distinct contrast to those in which health is good for many years. There is no satisfactory explanation of why a few patients belonging to group 4 have competent peripheral arterioles in certain tissues, whereas those in the retina are thickened. Therefore, much investigative work has still to be carried out to unravel these problems. The combined efforts of ophthalmologists, internists and pathologists have already made notable advances in the knowledge of these obscure diseases, and the results so far attained have been a great stimulus for these workers in different branches of medicine to continue their joint efforts.

SUMMARY.

Studies of the ocular fundi have played a significant part in distinguishing primary, diffuse arterial disease from diffuse glomerulonephritis. Observations of the normal and abnormal retinal arterioles during life have added to the knowledge of the physiology and pathology of arterioles throughout the body. Spasm of the arterioles may or may not lead to retinitis. When spasm co-exists with demonstrable histologic arteriolar lesions, retinitis and thrombosis result. Relaxation or intensification of

arteriolar spasm may explain the remissions and exacerbations that occur during the course of diffuse arterial disease. Such studies suggest the possibility that there are peripheral compensatory mechanisms when even serious arteriolar lesions exist. Retinal vascular changes are of practical significance in prognosis.

THE MAYO CLINIC.

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TABLE I.
SUMMARY OF CASES.*

Case	Date	Blood pressure	Heart		Retina				Urine			Renal Function	Comment			
			Pulse rate	Size, Grade	Electro-cardiogram	Periphetal sclerosis, grade	Sclerostis, grade	Retinitis, grade	Vasospasm, grade	Specific gravity	Albumin, grade			Casts, grade	Erythrocytes, grade	Blood urea mg. For each 100 c.c.
1.	12-5-23	160/115	74	1		1	1	0	1	1.018	1	1	0	16	45	Spasm of artery observed Thrombosis of retinal vein
	9-29-24	170/120	1				1	0	1	1.017	0	0	0	17		
	4-13-26	160/110	1		2	1	1	0	3	1.015	2	0	0		45	
	8-8-30	155/100					2	1								
		150/110														
2.	10-27-31	220/110	75	1-2	+	3	See text		1.008	2-4	1	4	28	30	Photograph of retina (Fig. 1) Biopsy of muscle (Fig. 3)	
	10-31-31	200/140	65						1.017						Died	
3.	2-1-32															Died
	3-23-32	150/105	110	1	0	2	0	3	1	1.009	4	0	2	222		
	3-25-32	120/90												222		
4.	12-28-25	180/100	100	1	0		0	0	0	1.009	3-4	1-3	1-2	50	50	Acute pericarditis Tonsillectomy Moderate edema Slight edema Died
	1-22-26	130/80								1.020						
	6-2-26	160/100	80	1		1	0	0	?	1.018	3-4	0-2	2	78	45	
	6-19-26	135/80								1.012	1-2	1	1	56	50	
										1.025						
	10-13-26	180/110	80	1		1	0	1	?	1.007	1-3	0-1	0-1	59	50	
	11-27-26	160/100	115	1	1	1	0	2		1.025				177		
	11-29-26	180/120													Died	

5.	9-28-22	145/90	96	1	0	2	1	0	1.015	1	0	0	6	Blood creatinine 7.2 mg. in each 100 c.c. Died	
	7-18-29	165/90	90	0	2	2	1	0	1.008	2	0	1			126
	6-20-32	220/110	90	1	0	2	1	0	1.011	2	0	1			
	6-27-32	150/90	90							2	0	0			118
6.	7-3-32												Well		
	7-16-18	120/80	72				0	0	1.029	0	0	0			
	7-9-24	130/80	72	0	0		0	0	1.024	0	0	0			
	11-17-30	210/130	75	1	0	2	1	0	1.003	0-2	0	0		32	
7.	7-21-31	170/100	68	1	2	1	0	0	1.015	0	0	0	16	Condition good Good health	
	7-25-32	160/90													
	7-25-32	200/130	70	1	2	1	0	0	1.027	0	0	0	30		
	5-19-28	205/160	85	0	0	1	0	1	1.001	0	0	0	15		
8.	4-23-31	140/115							1.028				Condition worse Photograph of retina (Fig. 8)		
	8-6-32														
	5-6-29	230/145	80	1	0	1	2	0	1.001	0-1	0	0		19	50
	6-6-32	260/160	80	1	2	2	2	1	1.031					32	
9.	12-21-31	260/175	95	2	2	2	2	0	1.003	0-2	1	0	18	35	Photograph of retina (Fig. 9) Condition improved
	175/125								1.016	2	0	0	45		
	12-30-31														
	3-18-32	220/160	85	1	2	3	2	2	1.005	3-4	1	1-4	38		
10.	5-4-32	190/130	90	1	2	2	2	2	1.035				80	Photograph of retina (Fig. 10) Died	
	5-21-32	140/90	120						1.011	3-4	1-2	1-4	232		
	5-21-32														
	5-26-32								1.019						

*Wherever the symbol zero appears it means that no abnormality was present, although an endeavor was made to detect one; wherever a space is blank, it means that a test, procedure, or examination was not carried out.

†Inverted T wave in deviations 1, 2 and 3.

‡Electrocardiographic changes present.

X.

THE PRACTICAL APPLICATION OF WITTMACK'S
THEORY OF PNEUMATIZATION.*

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Since Wittmaack first expounded his theory on the mechanism responsible for the pneumatization¹ of the temporal bone a great deal has been written on this subject. More and more proof has been brought out until now one can accept as fact the principles of the normal process of pneumatization which Wittmaack has set forth. In brief, these are as follows:

The mastoid process of the newborn child, when seen histologically, appears as a spongy bone showing fatty bone marrow filling the spaces between the bony trabeculae. The middle ear at birth is completely filled by a high embryonal type of myxomatous connective tissue which lies immediately under an epithelial covering. In the conversion of the embryonal tissue into its adult form there occurs a gradual shrinking and contraction of this tissue which eventually becomes the adult subepithelial tissue found everywhere underneath the epithelial lining of the ear and the mastoid cells. As soon as the child takes its first breath of air this myxomatous subepithelial tissue begins to grow from the antrum and epitympanic space into the adjoining marrow spaces located in the mastoid cells. In the course of time this embryonal connective tissue completely replaces all of the fatty bone marrow. This is the first step in the process of pneumatizing the temporal bone. As this embryonal tissue then undergoes its normal contraction to eventually become adult connective tissue it causes its epithelial lining to be drawn inward into the intertrabecular spaces. This constitutes the second step in the process of pneumatization. Finally, when the contraction of the embryonal

*From the Otolaryngological Department of Beth Israel Hospital. Read before the American Academy of Ophthalmology and Otolaryngology, Montreal, 1932.

tissue has been completed, the epithelium will be found to line the bony trabeculae within the mastoid process. Thus there results a series of spaces bound by the bony trabeculae and lined by epithelium under which is the adult form of connective tissue. These spaces all communicate with the antrum and are filled with air.

This is the normal process of pneumatization. It occurs in every one in whom no interference with the normal process is presented. This explanation of Wittmaack's meets with practically no opposition. On the other hand, his explanation of what occurs when the normal process of pneumatization is interfered with has been subjected to a certain amount of criticism by some observers.^{2 3 4} However, Wittmaack has been able to show by histologic study and has been substantiated by many observers along anatomic, clinical and roentgenologic lines of investigation, that the result of an interference with the normal process of pneumatization causes a failure of the mastoid process to become pneumatic.^{5 6 7 8 9} There are two main types of mastoid processes which will result from an interference with pneumatization: the infantile or diploic and the sclerotic mastoid process. However, depending upon the stage at which the normal pneumatization is interfered with, a mixture of types within the mastoid process may result.

If an uninfected foreign body, such as meconium, vernix caseosa or vomitus, enters the middle ear through the eustachian tube before the process of pneumatization has been completed, there will occur a disturbance of this process. The irritation caused by the foreign body on the embryonal type of connective tissue will produce a hyperplasia of the subepithelial connective tissue and prevent the normal contraction of this tissue. Consequently, the ingrowth of this subepithelial myxomatous tissue into the marrow spaces of the mastoid process will not be followed by any attempt at contraction due to the hyperplasia which has been set up by the foreign body irritation. The first step in the process of pneumatization therefore occurs, but it will not be followed by the second step, namely, the normal contraction and drawing in of the epithelial lining. This fibrous tissue which has replaced the fatty bone marrow will persist within the intertrabecular spaces, and in the course of time will be subjected to the osteoplastic

activity of the growing osseous trabeculae. There thus occurs a metaplastic ossification and there then results a sclerotic mastoid process.

Should this type of interference with the normal process of pneumatization occur after a certain amount of contraction of the subepithelial connective tissue has set in and a few pneumatic spaces have been developed, any further pneumatization will be stopped at that point and there will then result a mixed type of mastoid process showing partial sclerosis and partial pneumatization.

On the other hand, where a pathogenic micro-organism enters the middle ear and produces a suppurative infantile otitis media, the inflammatory lesion, after it has run its course, will be healed by an evidence of increased fibrosis in the inflamed tissue. This tendency to heal the suppuration by a production of fibrotic changes causes a very rapid contraction of the subepithelial myxomatous tissue. This then results in an enhancement of the contraction of the embryonal tissue. Consequently, if this type of infantile otitis should occur before the subepithelial tissue has grown into the mastoid process, such an ingrowth will never occur because of the rapid fibrotic contraction. There will then be, throughout life, a retention of the infantile type of mastoid process, namely, the spongy or diploic mastoid, because the pneumatization was interfered with before the first step in its development could take place.

If an infantile otitis should occur during the first stage of pneumatization, there would result a mastoid process which would show a combination of all three types, namely, one wherein there would be areas of cellular development, sclerosis and diploe.

I have accepted Wittmaack's explanation of the pathologic pneumatization as the only one which logically explains the presence of sclerosis and diploe in adult temporal bones. Only on this basis can one account for the hitherto apparent discrepancies in our explanations of chronic purulent otitis media. Since the sclerotic mastoid process is the result of a failure of the bone to become pneumatized rather than, as formerly believed, the result of a disease which converts a pneumatic into a sclerotic bone, this sclerosis should not be viewed as pathologic. That a sclerosis

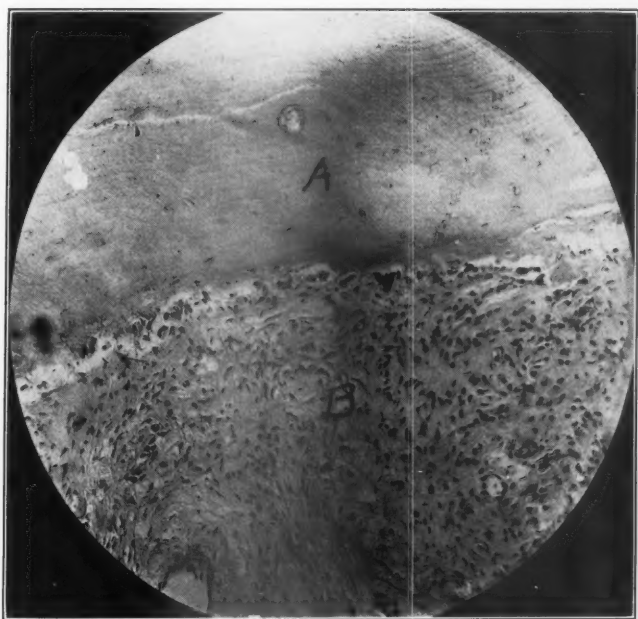


FIGURE 1.

A. Cortex of mastoid.
B. Dense connective tissue.

rarely, if ever, results from a chronic inflammatory lesion in a pneumatic bone has been demonstrated histologically by Wittmaack¹⁰ and Beck.¹¹ I also wish to add histologic proof of this. In one of the cases in my series, a child of seven, who had had a chronic otorrhea since the age of two, following scarlet fever, and on whom a radical mastoid operation was performed by me, the microscopic examination of the mastoid process revealed a normal cortex beneath which were bony trabeculae containing dense connective tissue. At no point in the section was there any sclerosis noted. In this we have evidence that where an acute necrotic otitis occurs during the course of scarlet fever in a temporal bone wherein normal pneumatization has taken place and wherein no surgical mastoiditis has developed, the cellular

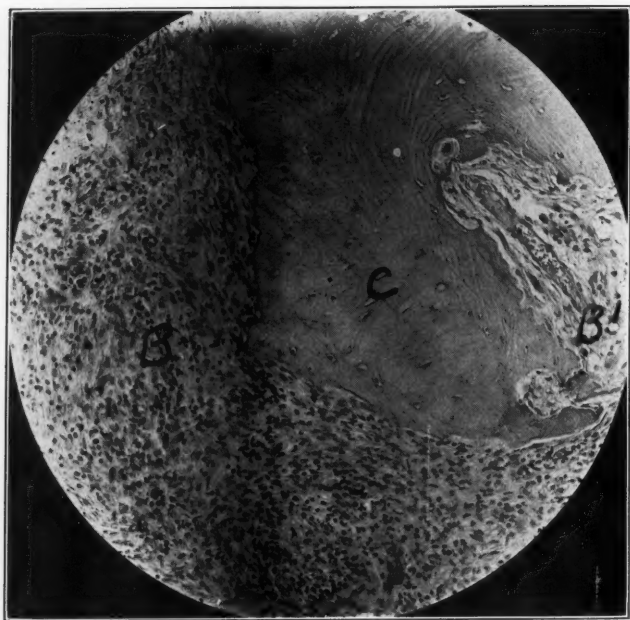


FIGURE 2.

C. Bony intercellular trabeculae.

B and B'. Dense connective tissue filling two formed cell spaces.

elements within the mastoid process which remain intact are not obliterated by any eburnation or sclerosis. Rather, the healing takes place just as anywhere else in the body, namely, by resorption of the exudate and replacement by connective tissue.

If we then take as our premise Wittmaack's teaching that eburnation or sclerosis found within a mastoid process is not pathology or the result of the pathologic condition present in the middle ear, epitympanic space and antrum, we must conclude that there is no necessity to surgically remove this eburnated or sclerotic bone. Rather we must view the disease for which we operate as being confined purely to the middle ear and consider it solely as a disease of the middle ear. In those cases wherein the chronic purulent otitis media is due to bone necrosis the gross

pathology always shows the disease as limited to the middle ear, attic and antrum. At operation, after the dense sclerotic bone has finally been entered, there is always found a small space represented by the antrum, from which the necrotic area can be seen extending into the epitympanic space and tympanum. Consequently with this in view, I have within the past two years modified the technic of the radical operation in cases of bone necrosis to suit the individual case. Where the entire mastoid process is found sclerotic, as determined by X-ray, the surgery is limited to the antrum and tympanic cavity and whatever area surrounding the antrum may be diseased. The sclerotic bone occupying the remainder of the mastoid process is left in situ. The technic of the operation is no different than that described in the textbooks with the exception of the use of a drill to go through the cortex into the antrum and to smooth down the bony walls and the limitation of the operative procedure to the middle ear, antrum and whatever area surrounding the antrum is found diseased. This leaves a very much smaller area to be epidermatized and consequently shortens the after-treatment.

The attempt to individualize the radical mastoid operation and to limit the extent of the surgery employed in cases of chronic purulent otitis of the dangerous type is meeting the favorable consideration of other otologists. Rassudov,¹² Alexander,¹³ Joseph,¹⁴ Babbitt,¹⁵ Smith¹⁶ and others advocate various procedures designed to limit the scope of the radical mastoidectomy, and have reported excellent results. The reason for the success of these measures is found in Wittmaack's teaching that sclerosis in a mastoid process is not a pathologic condition which requires removal. Of seventeen ears which have been operated upon in the manner I have described, seven showed complete epidermization and cessation of the discharge four weeks after operation; five had a similar result within five weeks after operation, two within six weeks, one within seven weeks and one within ten weeks. The remaining case, one wherein a labyrinthine fistula was found, still has a discharge after three months, due to a sequestrum, and will require further surgery.

Another factor in Wittmaack's work which must be considered is his teaching that an inadequate escape for epithelial debris will

result in the production of a cholesteatoma in a case of chronic purulent otitis media of the transformative type. Where a cholesteatoma is encountered as the cause of a chronic purulent otitis media, the same operative procedure is employed as previously outlined. The greatest percentage of cholesteatomas which came under our observation are secondary cholesteatomas and are the result of an ingrowth of squamous epithelium into the middle ear and antrum in an effort to replace the diseased tympanic mucosa with healthy epidermis. In a previous paper presented before this Society,¹⁷ I endeavored to show that the epidermis played an important rôle in the repair of chronic suppurative otitic lesions. Only in those instances where a free exit for the desquamated epithelium was lacking did a cholesteatoma develop. The secondary cholesteatomas are the result of inadequate avenues of escape for the desquamated epithelium in the attic and antrum. The retention of this so-called cholesteatomatous material causes pressure upon the surrounding bone, a rarefying osteitis and a resultant enlargement of the cholesteatoma tumor mass. Consequently, since the matrix of the cholesteatoma is in reality nothing more than epidermis, and since in the healing of a radical cavity the epidermis lining the membranous canal is utilized to line the radical cavity or a graft of epidermis is employed, I feel that it is not necessary, except where invasion into the endocranium has occurred, to remove this matrix when performing a radical operation for cholesteatoma. Rather, this matrix should be conserved and utilized to line the radical cavity. The healing of the cholesteatoma tumor and the prevention of its further growth will occur as soon as adequate drainage is afforded and a large opening made so that no epithelial débris can accumulate within the middle ear, epitympanic space or antrum.

In operating upon cases of cholesteatoma I have limited the radical procedure by entering the cholesteatoma cavity through the mastoid cortex, removing the accumulated epithelial débris within it, lowering the posterior meatal wall to an extent which will permit adequate drainage into the external canal, removing the diseased ossicles, where present, removing the polypi and epithelial débris in the attic, but retaining the cholesteatoma matrix everywhere. In five cases of cholesteatoma upon which this

procedure was followed two had a complete cessation of discharge with complete epidermization in four weeks, two in five weeks and one in six weeks. All these five cases have since been under observation, the shortest time for any one case being six months, and none have shown any signs of recurrence of aural discharge nor does the epidermis lining the radical cavity in these cases appear any different from that found in my cured cases of bone necrosis.

Following radical mastoid surgery, one of the greatest contributory factors toward poor results is the failure to produce a sufficiently adequate opening in the external auditory meatus. During the performance of the plastic operation on the posterior membranous canal wall, a resultant small meatal opening will permit the desquamated epidermis to accumulate within this cavity following complete epidermization of the cavity. The purpose of the radical operation is therefore defeated, since a cholesteatoma tumor mass will then result. Consequently, since this tumor formation will occur only if drainage is inadequate, it can be obviated by paying more particular attention to the size of the meatal opening during the performance of the plastic operation on the membranous canal.

In two of my cases such a secondary cholesteatoma resulted after radical surgery had been performed. In one instance several radical operations were performed and a recurrence of cholesteatoma took place each time. This case showed a complete atresia of the external canal. The operation was confined to re-opening the radical wound postauricularly, evacuating the accumulated epithelial debris and the performance of a plastic operation on the meatus and external canal which assured a large opening. This case gave a completely dry ear within five weeks and has been under observation for six months with no sign of recurrence. The other case was similar, except that the patient had had one radical operation prior to the one I performed. After following out the technic as outlined in the above case with the performance of a plastic operation on the canal to assure a wide opening, a complete cure has been obtained. This case has been under observation since for one year.

Another point which must be considered in connection with Wittmaack's theory of pneumatization is the extent to which the temporal bone may become pneumatized. We are accustomed to consider in our everyday practice that pneumatization is more or less confined to the mastoid process and the areas immediately surrounding it. We consider the finding of pneumatic cells in the zygoma, squama and occipital bones as not unusual, and in the performance of our simple mastoidectomies we endeavor to eradicate down to the inner table all cellular elements which we can find. Nevertheless, on closer consideration, we realize that pneumatic cells can occur in other portions of the temporal bone.¹⁸ Since the subepithelial tissue of the tympanic cavity and antrum normally grows into the marrow spaces surrounding them, this ingrowth of subepithelial tissue may take place into the marrow spaces of the perilabyrinthine area and the petrous tip if these spaces are in direct contact with and subjected to the pneumatizing influence of the tympanic mucosa. Where such a condition occurs, there will then result pneumatic spaces in the perilabyrinthine area and in the petrous apex. Consequently, a purulency within the middle ear, which in most instances spreads into the pneumatic cells within the mastoid process, may very well spread into the pneumatic cells of the petrous apex where such exist. The pathway of infection from the middle ear in such instances may lead into the petrous tip and may result in an empyema of the petrous apex which, in its development and pathology, differs in no way from an acute coalescent mastoiditis.¹⁹ Where an extensive pneumatization of the temporal bone exists, therefore, one must be on a constant lookout for an involvement of the petrous apex by a spread of the infection into it from the middle ear.

Finally, Wittmaack's teaching presents us with the following warning. Since the process of pneumatization is a continuous one from birth onward, the finding of myxomatous tissue within the mastoid process of an infant does not in any way constitute evidence of a disease of the mastoid process as a causative factor in the production of athrepsia, anhydrenia or dehydration.²⁰ The pathologic studies made in cases wherein mastoid operations have been performed for the relief of these infantile intoxications

must carefully differentiate between tissue which is the seat of an acute inflammatory edema and the normal myxomatous sub-epithelial tissue which will be found at all times at the end of the first stage of pneumatization.

CASE REPORTS.

1. S. S., female, age 21 years. Discharging right ear since childhood. Etiology unknown.

Otoscopic examination: Total defect in right drum. Epidermis covering inner wall. Probe inserted into attic brought down cholesteatomatous masses.

X-Ray examination: Right mastoid process sclerotic.

Operation: November 2, 1931, radical mastoidectomy. Cortex entered through spongy spot with a drill. Large cholesteatomatous cavity found occupying antrum area. Cholesteatomatous mass removed and matrix left intact. Posterior wall lowered. Middle ear entered. Ossicles removed. Epidermis on inner wall of middle ear and attic not disturbed. Panse plastic performed. Entire cavity packed with rubber dam and posterior wound closed.

Result: Cavity completely dry and completely epidermatized on December 4, 1931.

2. Y. F., female, age 48 years. Discharging left ear since infancy. Etiology unknown.

Otoscopic examination: Large marginal defect in posterior portion of drum.

X-Ray examination: Sclerotic mastoid process, right side.

Operation: December 5, 1931, radical mastoidectomy. Cortex entered through spongy spot with aid of a drill. Antrum opened and found to be small, its walls necrotic. The diseased bone immediately surrounding the antrum removed. Posterior wall lowered and middle ear thoroughly curetted. Remains of ossicles removed. Stacke plastic performed. Cavity packed with rubber dam and posterior wound closed.

Result: Ear completely dry and completely epidermatized on January 2, 1932.

3. J. R., male, age 14 years. Intermittent right aural discharge since age of three years.

Otoscopic examination: Large attic perforation. Foul discharge.

X-Ray examination: Sclerotic right mastoid process.

Operation: July 14, 1931, radical mastoidectomy, right side, performed as outlined in Case 1. Cholesteatomatous mass in mastoid process found. Matrix left intact.

Result: Ear completely dry and completely epidermatized on August 9, 1931.

4. A. R. F., male, age 25 years. Right chronic otorrhea since age of ten years. Etiology unknown.

Otoscopic examination: Large marginal defect in drum. Foul discharge. X-Ray examination: Sclerotic mastoid process.

Operation: September 29, 1931, radical mastoidectomy performed as outlined in Case 2.

Result: December 4, 1931, ear completely dry and epidermatized.

5. N. J. B., male, age 25 years. Chronic purulent otitis media since age of ten years. Several polyps had been removed from the middle ear at various times. Middle ear curetted in 1928.

Otoscopic examination: Large marginal defect in drum. Foul discharge.

X-Ray examination: Sclerotic mastoid, right.

Operation: December 26, 1931, radical mastoidectomy performed as outlined in Case 2. Polyps and cholesteatomatous masses removed from middle ear and antrum. Cholesteatomatous matrix left in situ.

Result: January 23, 1932, ear completely dry and epidermatized.

6. E. B., male, age 26 years. Left chronic otorrhea since infancy. Radical mastoidectomy performed in 1926. Ear continued to discharge ever since then with no remission.

Otoscopic examination: Marked atresia of external auditory meatus. Through small fistulous opening; polyp seen coming from old radical cavity.

Operation: November 17, 1931, revision of radical mastoidectomy. Incision made through old scar and old radical cavity exposed. Found filled with a large cholesteatoma. All cholesteatomatous material removed and matrix left in situ. Plastic operation performed on external auditory meatus to assure a large opening. Posterior wound closed and cavity packed with rubber dam.

Result: Complete cessation of discharge and complete epidermatization on December 28, 1931.

7. E. M., female, age 29 years. Chronic otorrhea since infancy. Etiology unknown.

Otoscopic examination: Large marginal defect in drum. Foul discharge.

X-Ray examination: Sclerotic mastoid process, left.

Operation: November 25, 1931, radical mastoidectomy performed as outlined in Case 2.

Result: Ear completely dry and completely epidermatized on January 22, 1932.

8. E. F., male, age 36 years. Right chronic otorrhea for ten years. Vertigo and nausea for a year and a half. Spontaneous rotary nystagmus.

Otoscopic examination: Small marginal perforation in attic.

X-Ray examination: Sclerotic mastoid process, right.

Operation: December 1, 1931, radical mastoidectomy as performed in Case 2.

Result: Ear completely dry and completely epidermatized on January 4, 1932.

9. P. W., female, age 13 years. Right chronic otorrhea since age of eight years.

Otoscopic examination: Large marginal defect in drum. Granulation tissue in attic.

X-Ray examination: Sclerotic mastoid process, right side.

Operation: July 7, 1931, radical mastoidectomy performed as outlined in Case 2.

Result: Ear completely dry and completely epidermatized on August 15, 1931.

10. E. H., male, age 44 years. Bilateral mastoidectomy at age of four years. Otorrhea followed scarlet fever. Left mastoidectomy at age of

eleven years and again at age of thirteen years. One year later, radical operation performed. At age of twenty-one years, left ear reoperated; both ears then dry. At age of thirty-two years, reinfection of left ear with persistence of discharge to date. Pressure pain over left side of head.

Otoscopic examination: Complete atresia of left external auditory meatus. There seems to be a false membrane behind which is an accumulation of cholesteatoma. Right side shows well performed, completely healed radical operation.

Operation: January 15, 1932, revision of radical mastoidectomy, left side, after manner described in Case 6. Cholesteatomatous material completely removed and matrix left in situ. Plastic operation then performed on external auditory meatus to assure a wide opening.

Result: Ear completely dry and epidermatized on March 1, 1932.

11. G. M., female, age 31 years. Since scarlet fever in infancy, had chronic left aural discharge.

Otoscopic examination: Large defect in drum.

X-Ray examination: Sclerotic mastoid process.

Operation: January 15, 1932, radical mastoidectomy as outlined in Case 2.

Result: April 2, 1932, ear completely dry and completely epidermatized.

12. M. S., female, age 9 years. Right chronic otorrhea for seven years. Etiology unknown.

Otoscopic examination: Large marginal defect in drum. Foul discharge.

X-ray examination: Pneumatized mastoid process with cell outlines indistinct.

Operation: February 5, 1932, radical mastoidectomy. Complete excitation of mastoid process in which cells seemed to be filled with fibrous tissue and some gelatinous material. No pathology in mastoid process until the antrum was reached when distinct bone caries was noted. After the posterior wall was lowered the middle ear was found to be filled with granulation tissue and necrotic ossicles. Stacked plastic performed on the external canal. Cavity packed with rubber dam and posterior wound sutured.

Result: Ear completely dry and epidermatized on March 19, 1932.

Note: This case is the one referred to in the text and the photomicrograph of the mastoid process is shown.

13. F. L., female, age 26 years. Bilateral chronic purulent otorrhea since infancy.

Otoscopic examination: Marginal perforation in right drum, posterior portion. Left drum entirely absent.

X-Ray examination: Both mastoids sclerotic.

Operation: April 26, 1932, bilateral radical mastoidectomy after manner outlined in Case 2.

Result: Right ear completely dry and completely epidermatized on May 20, 1932. Left ear completely dry and completely epidermatized on July 14, 1932.

14. E. N., female, age 30 years. Left chronic aural discharge for twenty years.

Otoscopic examination: Large marginal defect in left drum.

X-Ray examination: Sclerotic left mastoid process.

Operation: June 20, 1932, radical mastoidectomy performed as outlined in Case 2.

Result: Ear completely dry and completely epidermatized on July 18, 1932.

15. J. R., male, age 16 years. At age of ten years had right simple mastoidectomy performed. Has had chronic otorrhea ever since.

Otoscopic examination: Total absence of drum and ossicles. Polyp formation in middle ear.

Operation: December 26, 1931, radical mastoidectomy. After the incision was made through the old scar, simple mastoid cavity was exposed and found to be filled with a large cholesteatoma. Cholesteatomatous masses completely removed and radical operation performed. Middle ear completely curetted of all diseased mucous membrane, bone and polyps. Cholesteatomatous matrix left in situ. Plastic performed on external auditory canal.

Result: Ear completely dry and completely epidermatized on February 15, 1932.

16. S. L., male, age 52 years. Left chronic otorrhea for thirty years. Dizziness and instability for two years. Severe headaches, one year. Fistula test positive.

Otoscopic examination: Large marginal defect in drum with polyps in middle ear.

X-Ray examination: Sclerotic mastoid process.

Operation: December 19, 1931, radical mastoidectomy performed as outlined in Case 2. Fistula found in horizontal semicircular canal. Plastic performed on external canal.

Result: Discharge still present due to a sequestrum evidently coming from the inner wall of the middle ear and antrum. Bare bone felt through external canal. Since all labyrinthine symptoms have disappeared since the operation, no operative procedure is being undertaken to remove the sequestrum at the present time. It is hoped that it will come loose of its own accord.

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XI.

RHINOLOGIC ASPECTS OF TEAR SAC SURGERY.

EDWARD KING, M. D.,

CINCINNATI.

Disregarding any difference of opinion that exists between the ophthalmologist, who believes in extirpation of the lacrimal sac and the rhinologist, who believes in the physiologic restoration of the flow of tears, in cases of dacryocystitis, there is still some disagreement among rhinologists regarding the technic of treating this disease. Extirpation of the lacrimal sac is a comparatively recent operation, having been performed for the first time in 1862 by Berlin. Attempts to restore the flow of tears through the nose, however, date back to the days of Galen, when the operation consisted of boring a hole through the lacrimal bone and the use of cauterization to produce a fistula through which the tears were supposed to flow. Up until the sixteenth century it was believed that the tears came from the brain, until Stenson, in an excellent description of the lacrimal gland and the lacrimal apparatus, dispelled this idea. In 1913, Anal was able to wash out the tear sac and to probe the duct. In 1926, West, in a paper read before the New York Academy of Medicine, described an experience with 2,600 cases of dacryocystitis and 1,600 operations. West, in all his publications, has given credit to Toti for the modern surgical approach to this problem. Toti published in 1904 an article describing his operation and reported some cases. Contemporaneously with West in the description of the intranasal operation, Polyak and Halle published cases describing their operations and claimed priority to West. Neither of these men, however, has had the experience comparable to that of West's.

In discussing the subject of dacryocystitis it is most interesting to consider the etiology. On account of the fact that severe infections of the conjunctival sac, such as blennorrhoea, are not found in the lacrimal passageways and since trachoma of the lacrimal passageways is rarely reported, we can rule out the

infection coming down from the conjunctival sac as the causative factor in dacryocystitis. Therefore, we must hunt somewhere else for the cause. Ordinarily we look for it in the nose, and we assume infection comes either through the sinuses or up through the nasolacrimal duct. In spite of this assumption, it is difficult to establish a close connection between diseases of the lacrimal apparatus and diseases of the nose and sinuses.

A brief review of the anatomy is important, because of the variations in the structures and because of the relation to the surrounding parts. The embryology shows a groove running from the inner angle of the eye to the olfactory pit. The epithelium in the floor of this groove forms into a cord and sinks into the mesoderm. Later on this cord becomes attached to the ectoderm of the upper and the lower lid. The branch which goes to the upper lid is attached at the extreme inner angle of the lid, whereas that going to the lower lid is attached more laterally. At about the sixth week the upper branch moves outward until it is opposite the lower arm. The part of the lid between the nose and the attachment of the duct is pinched off and becomes the caruncle. The lower end of this duct eventually finds an attachment in the nose.

The bony lacrimal fossa is composed of the lacrimal bone and the posterior lip of the ascending process of the superior maxilla. The fossa may be shallow and broad, or it may be narrow and deep. The bony canal is made up of these two processes with the addition at its lowermost portion of a small process from the inferior turbinate.

The membranous portion of the lacrimal apparatus consists of the puncta, the canaliculi, the sac and the duct. The puncta is imbedded in the orbicularis muscle. The canaliculus also runs through this muscle.

Formerly it was thought that the nasolacrimal duct was the important factor in the physiology of the flow of tears. At the present time most authors agree that the canaliculus has most to do with this function. The canaliculus, on account of its muscular action, pulls the tears from the conjunctival sac into the lacrimal sac. The latter normally being collapsed, dilates in the act of

winking and because of its fibro-elastic coat is able to force the tears into the nose. The sac is firmly held in its bed by the tendo-oculi muscle. There is a very slight line of demarcation between the sac and the duct. The common puncta is found at the uppermost portion of the sac. In dissections of about forty-five heads the common puncta was found at the very topmost portion of the sac in a large majority of the cases.

The duct and sac at times are very irregular and on occasion meet at an angle. The mucosa of the sac and duct is thrown into folds so that it was thought formerly that valves were present. Some authors have described a valve at the juncture of the sac and duct and at the opening into the nose. At the present time most authors agree that the valves which have been described are merely folds in the mucosa. The sac and duct are surrounded by a large plexus of veins and communicate with the nose through a foramen in the lacrimal bone through which a small vein runs. The sac and duct have a very heavy fibro-elastic layer. As the duct approaches the opening into the nose the mucous membrane takes on the character of the nasal mucosa. The opening of the duct in the inferior meatus is normally found just beneath the attachment of the inferior turbinate and 15 mm. from its anterior end. According to Shaffer, the manner of opening of the duct in the nose varies, so that operations in the inferior meatus for drainage of the maxillary antrum will injure the duct if it varies from its position just under the attachment of the inferior turbinate. In my experience, this is very rare. I made a study of forty-five heads in the Anatomic Department of the University of Cincinnati, and in none of these specimens, consisting of ninety ducts, was there any variation from the normal such as described by Shaffer. At birth the duct is supposed to be entirely patent. However, at times a small membrane is present, which eventually ruptures spontaneously and which can be influenced to rupture by means of light pressure on the sac. Instrumentation in these cases is contraindicated.

The relation between the nasolacrimal passageways and the nose and accessory sinuses has been described by Onodi, who has published a volume with excellent photographs on this subject. Mosher in this country has shown by drawings and anatomic

specimens the relation of the lacrimal fossa to the anterior ethmoid cells. It is well known that the agger-nasi cell, when present, is found in the thinnest portion of the ascending process of the superior maxilla, and it is always in close relation to the nosalacrimal duct. When the ethmoid labyrinth is developed far enough forward to be in close relation to the lacrimal fossa, the uncinate process ends in a cell which has for its outer wall the lacrimal bone. The antrum is in very close relation to the duct. The bony wall of the duct mounds out into the antrum, and when the antrum is large this duct is seen very prominently on the anterior part of the internal wall, forming a recess in the antrum which is called the prelacrimal recess.

The pathology is simple. Stagnant secretion, which is the result of obstruction and swelling of the mucosa, becomes a fertile field for the growth of bacteria. The mucosa swells and meets the opposite layer. The epithelium becomes softened and sloughs off, leaving a rough area which produces an ectasia. Repeated attacks of inflammation and infection produce a chronic dacryocystitis.

The Bacteriology.—Some work has been done on this by West, who claims that twenty-four hours after the intranasal operation the conjunctival sac is free from the pathogenic bacteria. Other authors have not verified this completely, but say that the bacteria disappear after the clinical recovery takes place.

The diagnosis of obstruction and suppuration is not difficult. Syringing out the sac and looking for the fluid in the nose is a simple method of establishing the patency of the passageway. We must endeavor to establish the cause of the dacryocystitis. That means a careful study of the nose by clinical examination and accurate X-rays, in order to determine the relation of the disease to the nose. The use of opaque media with X-rays was first discovered by Ewing in 1909. Since that time a number of authors have employed this method of outlining the sac. A few years ago Dr. Mary Asbury and I were associated, and during that time we made some studies by means of X-rays and lipiodol, so that we believe the study of these cases with X-rays and lipiodol is of value.

The treatment is generally in the hands of the ophthalmologist. We must attempt, however, to get the ophthalmologist to co-operate with us in establishing the cause of the disease, because it is possible that some nasal condition may be the contributing factor. Rhese has reported twenty-five cases in which he found that disease of the ethmoid sinus was the sole cause of the dacryocystitis. He operated on these cases after they had had a long course of treatment by the oculist without results, and in all of them a cure was obtained. If this is so, then it is important for us to study the nose carefully in order to rule out any nasal condition which may be the cause of the dacryocystitis. The use of probes and slitting the canaliculi is the common method of handling these cases. It must be admitted, however, that the use of small probes does not obtain sufficient dilation to be of any value and that large probes injure the canaliculi and at times result in perforation of the sac and duct.

If an operation to restore the flow of tears into the nose is contemplated, it is important not to interfere with the function of the canaliculi because if they are injured there is no need to do any operation. With one canaliculus intact, it is possible to obtain a good result. With a drainage operation the results are far more promising if both canaliculi are intact. There are two types of operations: the intranasal, which has been sponsored by West, and the combined, which has been described by Toti and modified by various authors. These operations have two things in common. First, the removal of the inner wall of the sac, and second, an opening into the nose sufficiently large to fit the remaining wall of the sac. The objections to the combined operation consist in the scar which follows the external incision and the injury to the muscle. As far as the scar is concerned, there is no serious reason why it should be an obstacle to the operation. If the incision is made carefully and closed carefully the scar is of no consequence a few weeks after the operation. West has objected to the external incision because it injures the muscle. There are many instances in which tendons have been dislocated during operative procedures and returned to normal function following the operation. In the external frontal sinus operation the superior oblique

tendon is displaced and the eye returns to normal almost immediately following operation.

The objection to the intranasal operation lies in the technical difficulties encountered in performing it. Polyak, who has done a large number of these cases and who is a contemporary of West, states that the operation is very difficult. Kofler of Vienna has proposed that the operation be done through a hole in the septum in order to have a better exposure of the field. Some authors proposed that a probe be pushed into the nose and the operative procedure follow the probe. Almost everyone agrees that the procedure offers technical difficulties which sometimes are insurmountable. As far as the removal of the anterior ethmoid cells and middle turbinate are concerned, the procedure is the same in both operations. If the ethmoid labyrinth is developed far enough forward to be in close connection with the lacrimal fossa, it is necessary to remove the anterior ethmoid cells. If the nose is narrow it is important to remove the anterior end of the middle turbinate. In a wide nose it is a matter of judgment on the part of the operator. Any operation which contemplates less than the removal of the entire inner wall of the sac and the making of a hole into the nose large enough to fit the remaining wall of the sac is to be condemned. West, in his early work, had failures because he operated on the duct instead of the sac. Other failures he had after beginning the work on the sac he attributed to the fact that he did not remove the entire inner wall.

The combined operation has been modified by a number of authors. Depuy-Du Temps and Bourget have reported a large series of cases with excellent results. Mosher, in this country, has modified the Toti operation and simplified it by changing the incision and neglecting the suturing of the sac to the nasal mucosa. I have a moving picture of the Toti operation, as modified by Mosher, which reveals the technic followed by him.

The only way for us to know what results we obtain is to follow these cases closely. Unfortunately, I have been able to follow only three cases, and the results in these cases are as follows: The first one, a boy of 14, was operated on by the combined method seven years ago. At that time he had chronic suppurating dacryocystitis with a fistula, and at the present time it is possible

to pass a probe into the nose from the outside, and he is completely free of symptoms. The other two cases are in elderly women, who were operated on to relieve dacryocystitis preliminary to cataract operations. One of these patients has been under my observation for two years and the other one a little over one year. In both cases it is possible to pass a probe into the nose and they are free from symptoms. Mosher has shown a case which was operated on by the combined method seven years previous to the death of the patient. A postmortem was performed and the opening into the nose was found without any trouble.

It is possible that the opening of the sac and the drainage into the nose is sufficient to restore the function of the passageways even though the opening into the nose closes up. However, this is merely a conjecture and we can only determine by a careful study of these cases following operation the end results.

CAREW TOWER.

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XII.

IMPROVED HEARING IN FIXATION DEAFNESS: CASE REPORTS—COMMENTS.

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LOS ANGELES.

This paper is based upon several unusual observations in cases of progressive deafness without significant evidence of nerve involvement. These experiences, which are new to me, seem to establish that recovery of a useful degree of hearing may occur after fifteen to twenty years of increasing impairment. While the findings have been characteristic of stapes fixation, no attempt is made to prove that otosclerosis has been the cause. (See audiograms under "Case Reports.")

For the sake of brevity in presentation, case reports are appended; in each case bilateral progressive deafness with tinnitus, associated with no known infections, inflammatory or toxic cause, after increasing, despite treatments, for fifteen and twenty years, respectively, has shown striking improvement following the use of systemic measures. In each case report the treatment has been set forth, not as a definite method for treating such cases, but in order to render the report complete.

The cases suggest certain clinical and biologic considerations:

(1) The deafness followed tissue changes which altered size, shape or texture of bony or ligamentous structures near the oval window:

(2) Clinical evidence suggests gradual increase in degree of these tissue changes (see slides showing various changes in tissues);

(3) Bilateral incidence of this type of deafness suggests predisposition to local cellular or tissue changes;

(4) Bilateral incidence of this deafness points to systemic factors whose operation activates the process; some of these systemic factors are commonly associated with puberty and pregnancy;

(5) That attempts to alter the constitution of body tissues have been followed by change in long-standing ear conditions, is shown

by the onset of vertigo, nausea and vomiting, by cessation of tinnitus and by increased hearing acuity; coincident general tissue changes were seen in alterations of body weights, tissue and circulatory tensions, metabolic rates and blood pictures.

For over twelve years I have been trying to find, in physics and bio-chemistry, further information which would, directly or indirectly, facilitate study of these interesting but obscure problems. Researches in many fields show that, after nutritional and other environmental factors are altered, biologic changes occur which might explain the clinical phenomena presented. There is a tremendous amount of available data, which is obviously impossible to present in this paper. This data points unmistakably to cellular and organismal mechanisms, and to extrinsic and intrinsic factors, all of which take part in the tissue changes under consideration. These biologic changes and mechanisms are revealed to some extent by a few selected items.

CHEMICAL FACTORS.

I. Horticultural experiments¹ determined that, if a tree trunk be gashed, the natural process of healing by cell replacement may be arrested by allowing distilled water to flow over the wound margins. This suggests that a water-removable substance plays a part in the process of normal cell replacement.

II. Mathews² showed that bird muscle extracts oxygen from the air, and that grinding up the muscle increases the rate of oxygen extraction.

III. Kenyon³ showed that ground-up bird muscle, if washed with distilled water, ceases to extract oxygen from air; and that neither the residue nor the wash water extracts oxygen. Items II and III suggest that combination of water-removable substance and residue is necessary to the extraction of oxygen; considered in connection with Item I, definitely chemical processes seem to be concerned with cell division.

ELECTRICAL FACTORS.

IV. Buff, Matthews, Mueller-Hettlingen⁴ and others have studied current of action in living tissues; they found that, in living organisms, currents flow from older to younger, growing portions of an organism.

V. A green leaf,⁵ being partially exposed to sun rays, shows action currents, the radiated portion being electronegative to the shaded portion.

VI. Waller⁶ found that bruised portions of bird muscle are electronegative to unbruised portions.

ELECTROCHEMICAL FACTORS.

VII. Parsons⁷ found the center of a malignant tumor to be electronegative to the periphery. Beebe⁸ states: "The tissue of a tumor is probably typical and may be expected to have characteristics distinguishing it from normal tissue as sharply as various normal tissues may be separated from one another; . . . in its activities, tumor tissue forms so marked a contrast with other tissues it may be expected to show chemical peculiarities as well." He has reported chemical analyses of tissues confirming this concept. Other observers have confirmed such findings also.

Items IV, V, VI and VII suggest that, in communities of living cells, different levels of potential are manifested as action currents. Electrocardiograms exemplify such potentials, as do "nerve muscle" preparations in biologic and physiologic laboratory experiments.

POLARITIES.

VIII. Polycellular organisms manifest electropolarity. Lund⁹ found that trees show current of action, the growing tips of branches or of roots being electronegative to the older portions. By staining, he found that reversing polarity in the branch of a tree results in stopping the up-flow of sap. Experiments in which artificial reversals of their polarities were impressed upon certain living organisms showed that: *Bursaria* react by reversed beating of their cilia; *paramecium* react similarly, and sustained artificial reversal ultimately results in the development of an ostium at the caudal end; in the hydroid *obelia*, which has a polyp at one end, a hold-fast tail at the other, reversed polarity causes polyp to develop at the tail and the hold-fast at the polyp end.

These observations shed further light upon the nature of normal growth changes and some of the physiologic activities of living organisms.

MECHANICAL FACTORS.

IX. Gray¹⁰ observed that, when daughter-cells were separated after the first, second or third cell-divisions of a fertilized sea-urchin egg, each daughter-cell, so separated, develops into a full normal sea urchin; but not after the third cell-division. These findings shed light upon the nature of a cell mechanism concerned with the evolution of fertilized ovum into normal adult.

TIME-ENERGY FACTORS.

X. Loeb and Northrup¹¹ determined the average duration of life in eighty-seven generations of fruit flies raised under absolutely microbe-free conditions, but at different temperatures. They found that flies living at 30° centigrade lived less than half as long as those at 20°; those at 10° lived over eight times as long as those at 30°. The rate of living is apparently controlled by organismal mechanism susceptible of influence by heat. This recalls the chemical effects upon cell mechanisms exemplified in Items I and III.

CELL-REPLACEMENTS.

XI. Normally, individual body cells are presumed to survive for only a portion of the life of the body; on reaching the end of its normal existence, a body cell is supposedly replaced. The constancy with which facial, postural, statural, pigment and other individual characteristics persist over the span of fifty to seventy years seems to show that, however these physiologic cell replacements occur, they must be on a pretty accurately one-for-one basis; otherwise bodily or facial asymmetries and other similar alterations would show up in the course of a long life. This "one-for-one theory" involves assuming some control mechanism in the cell which is being replaced. That such a control actually exists is indicated also by evidence of its disturbance in conditions of endocrine imbalance which manifest dwarfism and gigantism; also in acromegaly.

XII. Cell growth *in vitro*¹² has been observed to go on indefinitely on homologous sera; also on body juices (a) from the immature animal, (b) from the mature cancerous animal; heating these juices to 155° produces no evidence of change in those from

the immature animal, but does change those from the mature cancerous animal, in that they no longer sustain cell growth. There are, evidently, substances in these juices essential to sustained cell growth in vitro; those from immature animal are thermostable at 155°; those from mature cancerous animal are thermolabile.

SEED MECHANISM.

A seed may be looked upon as a dynamic mechanism for building into organismal pattern matter particles available in the surrounding soil. In certain soil, when vegetable seed is planted, the building process does not occur at all; in other soil, plants grow, characteristic in form but of varying size, shape and flavor. Chemical analysis shows that their constituent particles differ as they are grown on different soils.

XIII. That the actual chemical constitution of vegetable tissues is susceptible of such chemical and physical changes is proved by many analyses. (See appendix B.)

XIV. In view of the foregoing, one may postulate probabilities of similar differences in animal tissues grown upon different foods. This is confirmed to some extent by many chemical analyses. (See appendix C, slides showing varying chemical constitutions of meats, oysters, etc., grown on different foods.)

EMPIRICAL.

Normally the human organism manifests abilities to restore many essential body structures; for example, fifteen years ago Dr. J. M. B.¹⁹ made a wide antrotomy, removing all overlying bone in a 1-year-old child, leaving the antrum "dished" wide open at the bottom of the operative field, which was healed over by soft tissue only. Fourteen years later X-rays showed no evidence of structural difference between this side and the unoperated side; Dr. H. S. M. did a complete simple mastoid operation through the old scar, finding anatomically normal cortex, cell structures, antrum and lamellar details. Another example of tendencies toward replacement of structural portions of the body is found following bone injuries. The removal of a transplant of bone sets into motion a series of local tissue changes which result

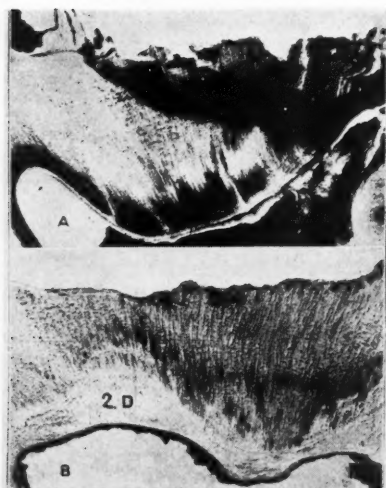


Fig. 6—A, active caries in a tooth before extra vitamin D added to diet; B, arrested or cured caries in another tooth resulting from the addition of much vitamin D to the diet.

Figure 1. Mellanby—J. A. M. A., Jan., 1931.

in complete restitution of bone corpuscles, osteoblasts, Haversian canals, vascular and lymph channels, duplicating the previous tissue conditions.

Mellanby²⁰ has shown that gross losses of dentine and enamel in teeth of calcium-deficient animals may be followed by similar restitution. (Fig. 1.)

ORGANISMAL MECHANISMS.

Such evidence of changing chemical constitutions of vegetable and animal tissues, and of restitutions of tissue losses, reveals cellular and organismal mechanisms concerned with cell chemistry and tissue replacements; in general, tissue restorations tend to follow normal anatomic pattern.

HALISTERESIS.

The withdrawal of calcium from bones and teeth, its circulation and redistribution in response to immediate needs, for instance,

in the nervous system, has long been postulated. The bony changes of rickets exemplify one of the clinically observed effects of this process; subsequent restorations of such withdrawals are observed clinically, and cytologic research has confirmed the correctness of this postulate.^{21 22 23 24 25}

SUMMARY.

The foregoing brief items bear significantly on this problem in that they show:

- (1) The existence of cellular and of organismal mechanisms;
- (2) whose operations involve chemical and physical factors;
- (3) which determine changes in size, shape and texture of tissues.

The deafness under consideration involves mechanical impairments of moving parts of the mechanism which conducts sound waves from environment to the perilymph.

RECAPITULATION.

1. The cases reported show, first, normal mobility; second, increasing immobility; third, decreasing immobility, of moving parts of this mechanism.

2. There are biologic as well as clinical reasons for attributing the loss of mobility to physical changes in certain tissues of the middle ear; and the same reasons explain the subsequent loss of immobility, signalized by the improvements observed in hearing.

3. Biologic studies have revealed some of the mechanisms of these tissue changes.

4. The clinical phenomena herein presented are explainable on this basis.

CASE REPORTS.

Mrs. M. S. Age 64, February 3, 1924. Deafness progressive in right ear for about twenty years, duration in left ear uncertain, but approximating in degree that of the right for several years; (later additional history elicited from others, left deafness had been noticeable much longer than stated—ten years or more); vague history of delicate health in childhood—measles, chickenpox, whooping cough—all before five years of age with no known sequelæ; nothing significant elicited concerning later childhood and adolescence, and nothing definite could be found out concerning family history. Married at 22, has two living children and one dead (early life, cause unknown); headaches for years not associated with gastro-intestinal symptoms; was told these were due to upper respiratory foci of infection (which produced no other symptoms). In 1909 nasal operations for cure of headaches, ineffective; gallstone operation in

1910; hysterectomy in 1914; operation for intestinal adhesions in 1921; states that right deafness was discovered in 1907 but that she noticed left deafness only in 1923; had many attacks of tonsillitis and lately increasing stiffness in knees. Had many examinations of hearing since onset in 1907, many courses of treatment, and ultimate diagnosis of "hardening of the ear bones" or "otosclerosis"; tinnitus has been a prominent symptom and has fluctuated in intensity with the deafness. Because of high blood pressure and joint troubles associated with acute tonsillar troubles, the tonsils were removed.

On March 28, 1924, general physical examination, blood count and urine were reported as "normal"; systolic level 190, diastolic level 120; the anterior end of the middle right turbinate was missing, the nasal septum thickened and irregular, but general conditions in the upper respiratory tract, teeth and gums were good; auditory canals and drumheads showed no significant changes; 128 d.v. heard 3 sec. longer in left than in right ear by air conduction, which was markedly shorter than bone conduction in both ears—bone conduction being markedly longer than tragus conduction in both ears; Weber not lateralized. Audiometer tests suggest high degrees of conduction impairment, more marked in the right ear; upper tone limit 5,000 d.v. in both ears, lower tone limit between 64 and 96 d.v. in both ears. In view of the failures experienced in former treatments, I advised her to try a long course of systemic measures to produce and maintain alterations of chemical body conditions; high fluid intake, minimum carbohydrates, alternating saturations with chlorides and citro-carbonates, each saturation lasting about two weeks, followed by interval of ten to fourteen days, during which iodides were taken for five to seven days. It was explained that no evidence of change was to be expected for many months; as this course of treatment was to take place at her home, she was advised to use some certain clocktick as a rough means of checking any change in hearing of either ear, and to report by mail after eight weeks or more; three and one-half months later she reported an "attack," severe headache, nausea and vomiting lasting several days; "in the midst of the upheaval my hearing came back suddenly and completely"; February 2, 1925, she was seen at my office, reported no recurrences of deafness and tinnitus; revision of audiometer was requested but her plans did not permit this examination and it ultimately transpired that no convenient opportunity presented. She has reported about once each year but has never found it convenient to permit audiometric study because of peculiar temperamental disinclination to pay any more attention to her deafness, "now that it is all right." In a letter written December, 1930, she states: "These last eight years (note, actually six years) have been very good ones for me through all sorts of weather; my knee gives me a little trouble * * * ; what a blessing it was that my hearing came back; my husband depended on me so many years to hear for him that it seemed a tragedy when I became deaf. * * *". In October, 1931, she reported a recent marked increase in deafness, associated with acute exacerbations of other troubles, viz: colitis, high blood pressure, disturbed rest, dyspepsia and occasional vertigo, which had resulted in general disorganization. Insuperable difficulties involved by her "colitis treatment," her "high blood pressure treatment" and other details prevented audiometric examination or other direct attention to her condi-

tion, and she proceeded to her destination, the desert. In March, 1932, she had recovered and I succeeded in obtaining the long deferred audiometric examination which showed marked improvement, as per lantern slide herewith presented. Three subsequent examinations in April and May confirmed these findings, as did clinical examinations of hearing.

Mrs. H. B., age 35, May, 1931. Fixation deafness over fifteen years, first noticed in left ear; similar tinnitus and deafness began in right ear year and one-half later; both parents died when she was eight years old and nothing definite can be learned concerning family history; she remembers hearing that she had had infantile colic, nutritional difficulties, chorea between 2 and 6 and generally poor nutrition and strength with frequent illnesses of unknown nature. Menses at 11 ushered in by pain and sickness, average duration six to seven days at two-week intervals; she grew very rapidly during the next two years, continuing to be slender and weak; recalls frequent bad colds from the age of 16, frequent epistaxis, no ear-aches or sore throat; also acute bladder troubles (pyelocystitis?), no gastro-intestinal troubles; laparotomy "because she was built too narrow," no pathology found, but "as long as she was open" appendix removed and she was told that "its neck was entirely too small"; had visual difficulties and nervousness for which glasses were worn and tonsillectomy was done with no apparent effect; wisdom teeth were extracted "to prevent ear trouble"; at 21 considerable orthodontia, including sawing off of protruding upper front teeth and substitution of pegged prostheses "to correct faulty occlusion"; increasing tinnitus and deafness followed, also beginning periods of constipation.

In social service and nursing work for several years; married at 26 at which time was menstruating five to six days at intervals of fourteen to twenty-eight days, with no intermenstrual discharge; one miscarriage about four months; ensuing pseudocyesis with milk formation at term; weight increased from 120 to 136 pounds, where it remained until last year. Deafness and tinnitus in both ears had reached maximal degree by the age of 30; during the past twelve years had frequent diagnostic studies and treatments at the hands of several excellent otologists, all of whom concluded that her trouble was otosclerosis. Audiogram in April, 1931, (herewith presented) consistent with such diagnosis; 128 d.v. conduction by bone much greater than by tragus in both ears; canals and membranes show no significant changes. History showed beginning arthritic changes during the past three years and occasional night sweating; for over ten years had carefully avoided protein food, using fats very moderately, carbohydrates heavily, and drinking very little fluid; upper respiratory mucosa was pallid but not hydropic; no obstruction or gross lesion in either naris; no significant pathology in pharynx or eustachian tubes; urine showed much epithelium, occasional pus cell; BMR -14 to -21; 4,200,000 red cells with color index over 1.0; 8,200 white cells, 51% polys., 20—, 35% small mononuclears, large monos., small trans. and eosin. 11-15%; systolic level 96-100, diastolic level 50-60.

She was given high fluid, protein, fat and very low carbohydrate diet; after two weeks, also alternating periods of chloridization and iodization—each saturation approximately two weeks followed by seven to ten day interval during which anterior pituitary and thyroid medication was used

occasionally for five to seven days; during the next six months her weight fell to 122 pounds, systolic level averaged 102-104, diastolic level averaged 70-80; no effects on hearing or tinnitus were noted but arthritic symptoms subsided and general health increased. During October and November 1931, reported frequent intermenstrual discharges; occasional discharges of mucus from the bowels occurred also; at Thanksgiving noted "momentary cessation of tinnitus" during which hearing seemed much clearer. During December and January these sudden interruptions of tinnitus with improvement in hearing were noted increasingly, longer and more frequently, and by February 1 these were occurring eight to ten times per week, each lasting from one to three hours. Audiogram of January, 1932, and of April, 1932, appended, show marked improvement; BMR, March 1, 1932, plus 4. (Fig. 2.)

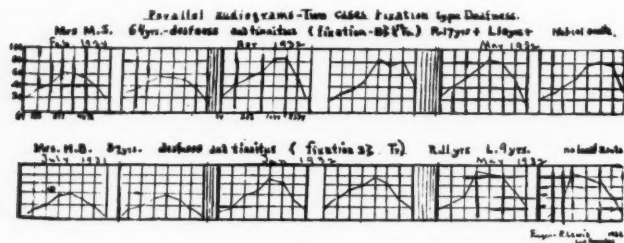


Figure 2.

APPENDIX A.

The therapeutic measures referred to represent attempts to produce and maintain chemical alterations in body tissues, as a possible means of effecting physical changes in tympanic structures whose mechanical operations had been interfered with. I am not attempting to show that these hearing impairments were due solely to stapes fixation, or that otosclerosis was the cause, or that the treatment was solely responsible for improvements which occurred; these and other details are relegated to some other occasion. This paper contains only two items: the clinical fact that recovery of useful hearing occurred in two cases after years of increasing fixation deafness, and the suggestion of a new approach in studying the problems of fixation deafness. The measures used in each case are set forth, not as definite methods of treatment, but, primarily, in order to render the reports complete. These reports, incidentally, serve to introduce an analogy between the changes which take place as matter particles organize

into cell form and crystal form. This, in turn, permits speculation as to whether something similar might be occurring in tissues in the course of changes observed clinically. (Fig. 3.)

No scientific reason is proposed for believing that what happens as particles organize into crystal bears upon what happens as particles organize into cell. The gossamer* of imagination dragging in such an idea is weak enough to safeguard against introducing into these deliberations any gross misconceptions. The exactitude with which the cell determines the distribution of incoming food particles according to its own individual structural pattern resembles that which characterizes the disposition of matter particles entering into the structure of the crystal. Sodium-ammonium-racemate occurs as oppositely symmetrical ("enantiomorphous") crystals of identical chemical constitution, the D crystal being the (Fig. 6) mirror image of the L crystal.

*Spun out of many unrelated bits: definite influences initiate morphologic changes whereby bacteria are transmuted into spore, filterable and non-filterable forms; normal mature epithelial and interstitial cells go over into abnormal forms (cancer and sarcoma) manifesting many of the characteristics of immature stages of their former selves; the structural pattern of yeast and other cell changes as they grow on different media (Fig. 4); X-ray study shows definite diffraction patterns in protoplasm resembling those of crystal; Crile has produced, in vitro, so called "synthetic cells," which undergo fission, consume oxygen and give off carbon dioxide; these might be considered as "crystals" which behave in some ways "as if they were living"; apparently as a result of changes in media, in spruce, lathyrism and pernicious anemia erythrocytes are seen to go over into abnormal forms—poikilocytes, macrocytes and microcytes; and these forms are again succeeded by normal erythrocytes when other changes in media take place; bone cells take on abnormal growth activities under certain inflammatory conditions affecting bone and periosteum (exemplified in "periostitis osteoproliferans"); in racemic solution of tartaric acid, the penicillium glaucum thrives on the dextrorotatory molecules, but when these are gone it is unable to survive on the laevorotatory molecules remaining, although these have the same chemical formula as the dextrorotatory molecules (Fig. 5). Claude Bernard believed that the metabolic condition of a cell depends on the composition of its medium; Carrel states "the rate of multiplication of fibroblasts (in culture) was found to depend on the concentration of certain substances in the pericellular fluid" * * * "fibroblasts, monocytes and epithelial cells modify their anatomical characteristics as well as their metabolic activities according to the composition of the medium." * * * "It is obvious that all metabolism and morphology depend on qualitative and quantitative characteristics of the humors." (Cowdry's Spec. Cytol.)

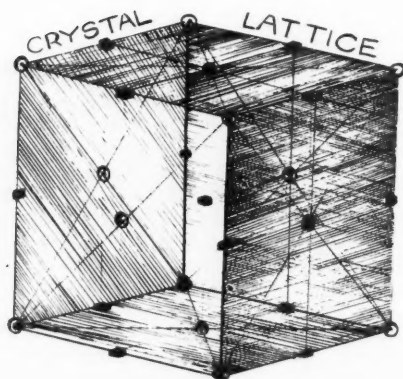


Figure 3.

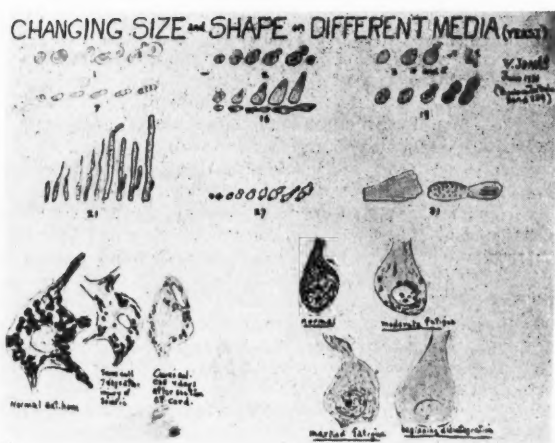


Figure 4.

In process of cooling, a mixed solution reaches the point at which crystallization occurs, with precipitation of both forms of crystal. Crystals are readily redissolved by adding water and heating, whereupon the solution may again be allowed to cool. If now a single D crystal be dropped into this cooling solution before the point of crystallization is reached, precipitation of D crystals only

ensues; if a single L crystal be dropped into it, only L crystals are precipitated. "Polarizations," initiated by the crystal inoculating the solution, determines such control of precipitations.

The cell is known to possess unique properties (i. e., some agencies or mechanisms) whereby its incoming particles normally continue, in accordance with chemical and physical laws, to dispose themselves in conformity with its peculiar structural pattern. How far abnormal conditions may disturb such disposal of par-

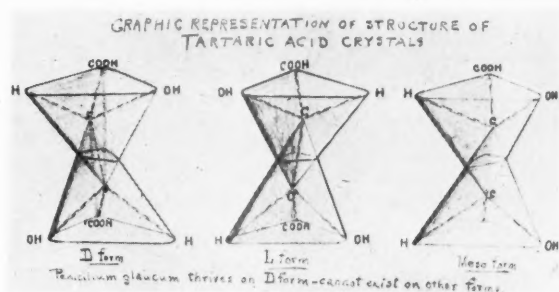


Figure 5.

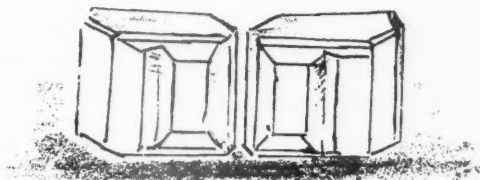


Figure 6. Enantiomorphous Crystals—sod-ammon-racemic.

ticles, and with what varying results, might repay investigation. On stimulation, during embryonic and immature stages,* cell energies are devoted solely to activities of growth; after maturity, solely to activities of function. Mutations of this kind suggest the existence and operation of some intracellular agency which

*When cell content of glutathione is highest, velocity of oxidation is highest, electrical cell output is highest. (Lund, Gray, Matthews, Muller-Hettlingen, et al.)

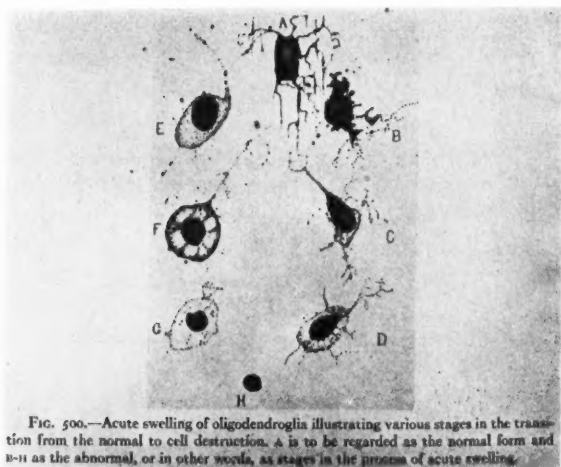


FIG. 500.—Acute swelling of oligodendroglia illustrating various stages in the transition from the normal to cell destruction. A is to be regarded as the normal form and B-H as the abnormal, or in other words, as stages in the process of acute swelling.

Figure 7. (Cowdry Cytology)

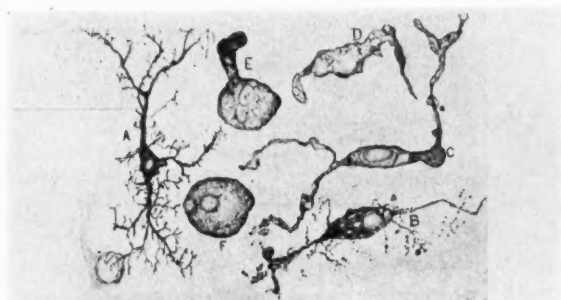


FIG. 503.—Stages in the transition of normal microglia (A) to compound granular corpuscle (r) from the human brain in the vicinity of a glioma. (After Penfield, 19250.)

Figure 8. (Cowdry Cytology)

itself undergoes gradual alteration as the cell "matures." This agency will bear closer consideration in the course of detailed studies of neoplastic, otosclerotic and other tissue changes.

Otosclerosis.—Between the fifth and tenth years of life the labyrinth capsule of certain individuals manifests the abnormal

bone changes of "otosclerosis"; these are in no way associated with health; they neither result from illness nor impair the health of the individual, directly or indirectly, as do toxic or infectious processes. Otosclerotic bone changes show reversions to immature and embryonal characteristics, among which is the devotion of cell activities almost exclusively to growth; in this respect otosclerosis might be described as "anachronistic" rather than pathologic. Clinical manifestations of otosclerosis are confined to evidences of mechanical effects of abnormal bone growth. In approximately similar locations certain neoplastic bone changes (exostosis, hyperostosis, osteoma) are accompanied by certain other effects—local pressure, pain and soft tissue changes. Otosclerotic tissues do not tend to break down; these neoplastic tissues do; the otosclerotic process speeds up during the physiologic changes concerned with puberty and pregnancy; these neoplastic processes do not.

Processes in which the structural pattern of a cell undergoes change (Figs. 7 and 8) may be compared with processes in which the structural pattern of a crystal undergoes change. The matter particles of calcium carbonate organize into rhombic crystals of "aragonite" below the temperature of 95°, into monoclinic crystals of "calcite" above that temperature. (Fig. 9.) Both of these crystals are of identical chemical formula, yet entirely different shape, size and structural pattern.

Whether as "living crystals" or as "synthetic cells," what Crile has reported (Fig. 10) might come to bridge a gap between these two kinds of organization.* Not only extraneous factors (temperature or pressure in the case of crystals, hormone or trophic influences in the case of cells), but also intrinsic factors (chemical constitution in the case of crystals, self-contained agencies or "catalysts" in the case of cells), influence organization processes of both crystal and cell formation. The outstanding and incomparable difference between these two kinds of organization of

*Suggestions of some such concept in approaching the general problems of crystal and cellular organization are found in researches during the first half of the last century; those of Pasteur, LeBel, Van't Hoff, Hartley, Baly, Desch, et al, led to "stereochemistry" and "spectrography" of modern science.

Below 94.5° Calcium Carbonate Crystallizes above

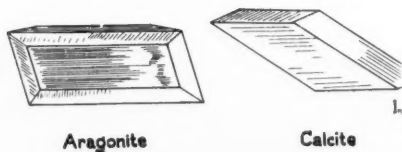


Figure 9.



Figure 10. Autosynthetic Cells (Crile).

matter particles lies in the fact that the identity of particles constituting a crystal remains unchanged, whereas the process of "living" entails an unending series of changes in identity of the particles which actually constitute the cell. This difference involves cell environment as a constantly varying factor, responsible for the constantly changing constitution of the cell.

The fact that otosclerotic cell activities are increased during puberty and pregnancy sheds some light on the nature of the abnormality. Some intracellular agency determines the direction of cell activities—at one stage, predominantly to growth—at

another stage, to other activities; otosclerotic cells act as if, in them, this agency were: (1) in the state normally found during embryonic life, at which stage such cells are "growth-minded"; (2) particularly stimulable by something whose concentration in the body is ordinarily increased during puberty and pregnancy.

Microscopic search through many specimens* fails to reveal details of bone cell structure—in lattice or other features—whereby one might attempt to differentiate "normal bone," "otosclerosis," "osteogenesis imperfecta" or "osteodystrophia fibrosa." Yet it seems not only possible but even probable that such structural differences do exist, just as do patterns which have been found to be peculiar to various crystal forms. On the basis of such a theory of protoplasmic or structural mutations, tissue changes such as those found in otosclerosis† may come to be looked upon from a new viewpoint; and the clinical experiences herein reported might serve to suggest new approach to studies of otosclerosis. Such a concept—the possibilities of peculiarities in structural pattern of living cells analogous to those found in crystal structures—is worthy of searching investigation. Differences in form of bony tissues suggest equally studious consideration of polymorphism found in phosphorus, calcium and sulphur, such essential components of bone.

APPENDIX B.

IODIN CONTENT, PARTS PER MILLION, DRY.

Weston ¹³	S. Carolina	California	Oregon
Asparagus	574.5	12.0	...
Green Beans	329.0	...	29.0
Spinach	1178.5	26.0	19.5
Beets	357.0	8.0	...
Carrots	466.0	8.5	2.3
McClendon ¹⁴ (See also ^{15 16 17})			
		Minnesota	Maine
Wheat		1.0	9.3
Oats		10.0	175.0

*Including those kindly furnished by Doctors Fowler, Meyers, Weber, Wittmaack, Fraser, Neumann and others, to whom I desire to express my most grateful appreciation and thanks.

†Also those of fibroma, osteoma and other neoplasias.

(Further data which had been assembled was unfortunately lost the first week of April, 1932. The foregoing, however, suffices to make the necessary point—that actual chemical differences are determined by differences in chemical constitution of the soil upon which a plant is grown.)

APPENDIX C.

Hunziker¹⁸—Iodin parts per 100 k. g. lv. wgt.:

	Live Weight
Simmenthaler	0.86
Shorthorn	1.60
Braunvieh	3.47
Norweger	2.36

Peterson, University of Wisconsin (lost), dried samples:

	Iron	Mang.	Cop.	Iodin	Cal.	Phos.
Beef Liver	294.0	8.7	75.7			
Calf Liver	203.0	12.0	164.4			
Hog Liver	800.0	12.2	20.8			
Oyster	760.0	34.0	95.0	16,000.0	4,400.0	6,200.0

Von Fellenberg—(Lost), fresh samples:

	Iron	Mang.	Cop.	Iodin	Cal.	Phos.
Beef Liver	83.0	2.5	21.5	19 to 87		
Calf Liver	54.0	3.2	44.1			
Hog Liver	250.0	3.8	6.5			
Oysters	76.00	34.0	9.5	1,600.0	440.0	620.0

Remington—(Lost), skimmed milk parts per million:

	Dry Matter %	Cal.	Phos.	Iron	Cop.	Iodin
Newberry, S. C.	7.59	15,000.0	7170.0	18.0	2.09	505.0
		14,800.0	7240.0	24.0	2.08	500.0
Greenville, S. C.	7.55	18,900.0	6330.0	13.0	2.02	840.0
		18,500.0	6400.0	53.0	1.96	862.0

Robinson & Huffman—(Lost), 100 samples, blood of normal mature cattle:

	mg. per 100 c.c.	mm.
Phosphorus	3.00—8.99	0.97—2.90
Chlorine	294—357	82.9—100.6
Calcium	7.7—14.7	1.93—3.68
Carb. Diox.	41.4—75.8	18.6—34.1
Magnesium	0.31—3.08	0.13—1.26
Potassium	16.4—41.3	4.2—10.5

Effect of changed diet on blood of calves, four animals:

	P	Ca	P	Ca	P	Ca	P	Ca
Jan. 15	7.26	9.5	7.14	10.8	7.18	11.4	6.31	11.3
25	8.68	12.5	6.94	13.0	8.33	12.6		
Feb. 15	8.12	12.7	7.10	13.0	7.35	12.1	5.89	11.6

Forbes & Swift—(Lost), iron content of beef, per cent.

No.	Iron	Nitrogen	Protein (Nx6.25)	Ether extract	Moisture	Mg. iron per 100 gm. of protein
No. 1	0.0024	3.24	20.25	6.05	72.70	7.9
No. 2	0.0025	3.55	22.19	3.48	74.65	11.3
No. 3	0.0025	3.29	20.56	6.39	72.02	12.2
No. 4	0.0025	3.17	19.81	7.13	72.84	12.6

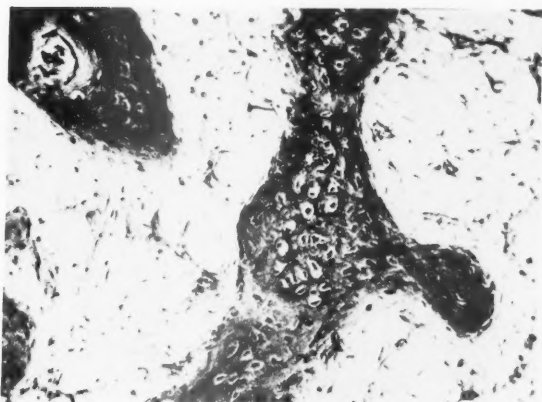


Figure 11. M. Weber. Normal fiber bone.

APPENDIX D.

Studies of characteristic differentiating normal and various abnormal bone tissues have engaged the attentions of investigators for years. No attempt is made to consider details of the work which is going on in this field; it is being well done and will doubtless attain successes worthy of all the efforts which have been put into it. Through the kindness and courtesy of Drs. Moritz Weber and E. P. Fowler, I have been able to examine many bone preparations from their collections, in addition to those which I have gathered in the past from the collections of Neumann, Fraser, Witmaack, Meyer and others. Careful search has failed to reveal, in any of the otosclerotic slides I have examined, the kind of sections I have sought—sections thin enough to reveal individual bone cell peculiarities and individual bone structure peculiarities.

Characteristics of individual bone cells are seen in the cuts of normal bone herewith presented. (Figs. 11 and 12.)

APPENDIX D.

Weber's section of normal bone in polarized light may well be studied side by side with his section of osteodystrophia fibrosa in



Figure 12. M. Weber. Normal bone-polarized light.



Figure 13. M. Weber. Osteodystrophia fibrosa. Bone cells in polarized light.

polarized light (Fig. 13), as an example of differences in structural pattern. (Figs. 12 and 13.)

Weber's section of osteodystrophia fibrosa and of osteogenesis imperfecta give glimpses of equally striking differences, both in

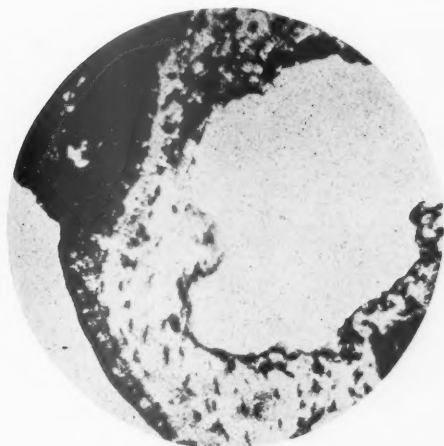


Figure 14. M. Weber. Osteodystrophia fibrosa. Bone cells.

bone cell and in structural pattern. Certain areas show particularly well their characteristic peculiarities. (Figs. 14 and 15.)

Studies of otosclerosis, in ultra thin sections, both by ordinary and polarized light, offer prospects of value in the matter of determining similarities and differences with respect to other abnormal bone changes—in the light of similar studies of both normal and abnormal bone cells and bone structure patterns.

The clinical experiences set forth point to (1) the necessity of patient, systematic studies of the biochemical aspects of normal and abnormal bone cell changes and of means for altering them *in vivo*; (2) to the important significance of other clinical conditions and changes found in association with such changes, and (3) in the course of general systemic attempts to change body chemistry conditions, to the significance of changes in blood picture, metabolic rate, body weight, tissue tensions, blood and pulse pressures, as presaging other tissue changes, in the course of which those contributing to deafness and tinnitus may undergo improvements commensurate with what has been observed in the cases reported.

1154 ROOSEVELT BLDG.



Weber fiber-bone, osteog. imperfecta

Figure 15.

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XIII.

THE USE OF THE AUTHOR'S IMPROVED BRONCHOSCOPE AND ESOPHAGOSCOPE IN DIAGNOSIS AND IN THE REMOVAL OF FOREIGN BODIES.*

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The developmental accomplishments of bronchoscopy and esophagoscopy have achieved an enviable place in our diagnostic armamentarium, and in the removal of foreign bodies.

Since the advent of Bozzini's ingenious speculum for the first visualization of the larynx, in 1804, instruments for the diagnosis and treatment of affections of the upper tracheobronchial tree and esophagus have been submitted to steady progress and improvement.

The early bronchoscopic and esophagoscopic tubes contained no form of illumination and the light was thrown into the tubes by means of a reflector, such as a hand or head mirror, or projected from a headlight, such as devised by Kirstein.

At the present time, the two outstanding types of instruments employed in bronchoscopy and esophagoscopy are, first, instruments wherein the illumination is proximal or in the handle, and second, instruments that are distally illuminated.

Bruening and Kahler provided the popular instruments bearing their names, employing proximal illumination or with the lighting system contained in the handle, and fitted with extension tubes in order to attain the required lengths in the various tubes to be used.

It was Einhorn, in 1902, who devised an esophagoscope, the side wall of which contained a channel running throughout its length for the insertion of a light carrier, such as is in use today and known as distal illumination.

*Read before the Mid-Western Sectional Meeting of the American Laryngological, Rhinological and Otological Society, Kansas City, Mo., January 21, 1932.

Chevalier Jackson, in 1904, used the tube of Killian, with the distal illumination of Einhorn, in a bronchoscope. He also provided an auxiliary drainage canal for these tubes. For the introduction of the tubes, Jackson devised a split spatula, patterned

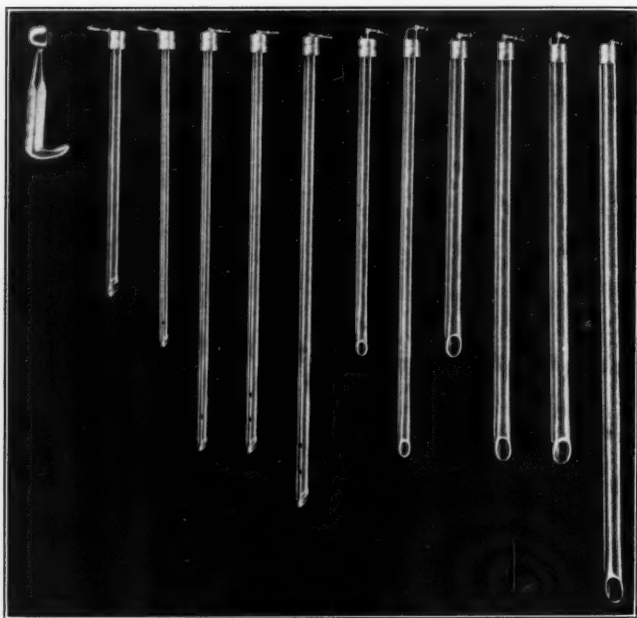


Fig. 1. Showing an assortment of esophagoscopic and bronchoscopic tubes, various sizes and lengths, and universal handle. The handle fits all tubes.

after the device of Killian, but separable, so as to avoid injury to the mucous membrane.

Since the development of distally illuminated instruments, particularly in America, there has been a constant clinical comparison as to the relative merits or superiority of the distally illuminated with those of the proximally illuminated instruments.

While it manifestly is a fact that with the proximally illuminated instruments one obtains greater projection beyond the distal

end of the tube, the handicaps or shortcomings of this type of illumination must be seriously taken into consideration.

Among these disadvantages or objections are that the longer the tube in a proximally lighted instrument, the greater the loss of light at the distal end. The fact is that in a proximally lighted tube, one must work in his own light, so to speak, or in front of the light, when instruments are inserted into the tube, producing blind spots or obscuring essentials at the distal end.

It is well known that oblique illumination presents a better view or perspective and more information is revealed than when the source of light falls flat on the field under observation. Oblique

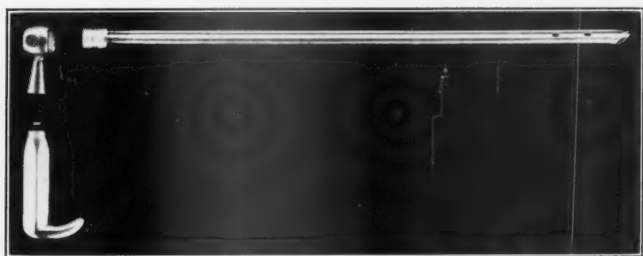


Figure 2. Detail of the separable universal handle, together with one of the bronchoscopic tubes, before assembly.

illumination is not obtained with any other type of instrument but those that are distally lighted.

In the proximally lighted instruments, the handles are heavy, clumsy or cumbersome and necessarily so, in order to house or contain the lighting system. Some types of proximally illuminated instruments, in addition to being heavy and unwieldy, are complicated, due to the many adjustments made necessary in order to properly focus the light into the tubes in the proper place.

In the distally lighted instruments, these objections do not present themselves, because of their simplicity of design and their minimum of weight.

The proximally lighted instruments, such as the Bruening and Kahler types, are directly introduced into either the trachea and bronchi or esophagus.

In the introduction of the tubes devised by Jackson, a laryngoscopic spatula is made necessary to expose the larynx. This laryngoscopic spatula is made separable, and through this spatula the necessary bronchoscopic tube is introduced, after which the laryngoscopic spatula is dis-assembled and removed, leaving the bronchoscopic tube in place. If, for any reason, the withdrawal of the bronchoscopic tube should become necessary, the laryngoscopic spatula is again utilized, in order to expose the larynx, and the same technic of introduction of the tube must be employed.

Curiously enough, this equipment, in the nature of the tubes, handles, electrosopes and means of introduction, has undergone very little change or improvement since about 1910.

Experience with the various instruments previously described revealed their various shortcomings and suggested the requisites of an ideal equipment which to me seemed most essential.

1. Simplicity.
2. Ease of introduction and re-introduction.
3. Lightness of weight.
4. Ease of assembly.
5. Adaptability.
6. Durability.
7. Large lumen or working space.
8. Safety.

With these essentials, it was not long before it was realized that only with a distally illuminated instrument could freedom from clumsiness and weight be obtained. This realization, coupled with the superiority of oblique illumination, made the decision in favor of distal illumination a certainty.

In the four years spent in the development of these improved instruments, many ideas and suggestions have been investigated, and at the present time they represent the results of a careful and prolonged study of the requirements, eliminating the shortcomings or objectionable features of previous instruments, and possessing improvements which, in my opinion, more closely approach the ideal of perfection, for which we should continue to strive.

The simplicity of these improved instruments render bronchoscopy and esophagoscopy a more simplified procedure and with comparative ease. There is no tiresome lifting up or strain, for

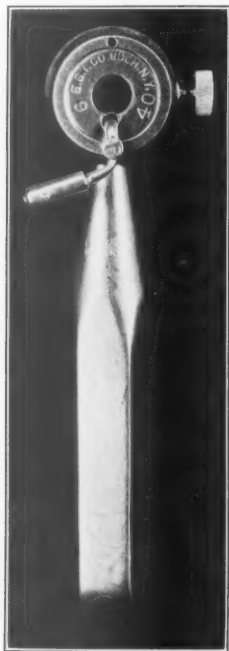


Fig. 3. Detailed close-up, of end view of handle and bronchoscopic tube when assembled, with light carrier in place and ready for use. The small hole at the top of the handle is for the light carrier pin. There is also a small hole below for the same purpose. The head of the tube can be rotated in the handle to any position, depending upon whether the tube is to be introduced either in the upright or recumbent position. The knurled screw at the right locks the head of the tube in the handle. The handle can be quickly attached or detached, to or from the tube, at will. Each bronchoscopic and esophagoscopic tube has the size and length stamped thereon, likewise the light carrier.

introduction, as is necessary when using other types of instruments, such as the Bruening. There is no clumsy or heavy handle, nor is it necessary to employ a laryngoscopic spatula or any type of accessory equipment in order to introduce any of the tubes.

The bronchoscopic tubes are rigid, and are made separate from the handles. They will not bend nor can they be dented by the teeth of the patient. The bronchoscopic tubes are similar in design to those of Chevalier Jackson, except at the distal end there is no acute ridge or flange, and no auxiliary drainage tube is employed, which would enlarge the outside diameter.

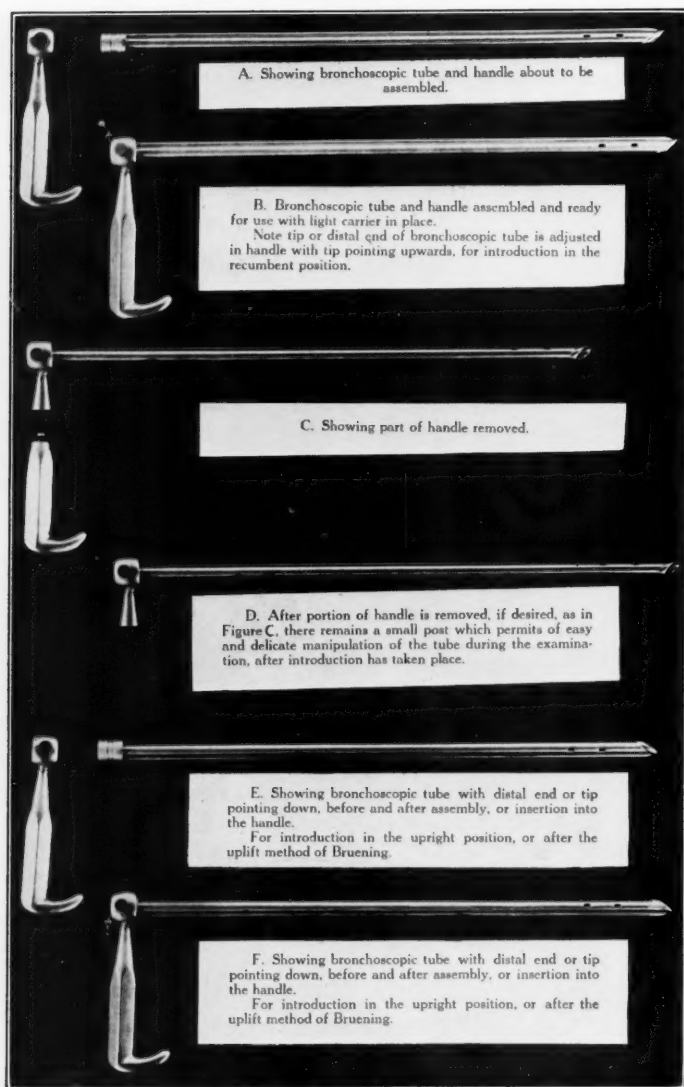


Fig. 4.

The proximal end consists of a circular head, which fits into a detachable, interchangeable and separable handle. Because of the circular head at the proximal end, the bronchoscopic tubes can be rotated in a complete circle in the handle. The same principle is incorporated in the esophagoscopic tubes.

The esophagoscopic tubes are made oval, which affords of easy introduction and a larger working space.

The handles are separate from the tubes and each handle fits all tubes.

By having the tubes separate from the handles and interchangeable, and the fact that the tubes can be rotated completely in the handle, makes possible the adjustment of the tubes in the handle, with relation to the distal end, depending upon whether or not the tubes are to be introduced in the recumbent or upright position.

The tubes are locked in the required position in the handle by means of a small thumbscrew. In the circular head of the handle are holes for the fixation of the light carrier pins, after the tubes and handle have been assembled.

The handle is so constructed that after introduction has taken place, the handle can be quickly removed, if desired, leaving a small post for the delicate control and guidance of the tube during the examination or withdrawal.

For introduction, the proper sized bronchoscopic or esophagoscopic tube is inserted into the handle, together with the light carrier, and locked in the required position by a small thumbscrew on the handle, provided for that purpose.

For introduction in the recumbent position, the distal end or tip of the tube should be pointing up, and in the upright position the distal end pointing down.

When the tube and handle are assembled, they are at an angle of 90° to each other, thus forming a right angle.

The distal end of the tube is inserted forward, in the midline, over the base of the tongue, and if a bronchoscopic examination is being made it is advanced until the lip or distal end of the bronchoscopic tube engages the tip of the epiglottis, which is held up by a gentle pull or pressure downward on the handle, which elevates the distal end, exposing the larynx and the landmarks contained therein.

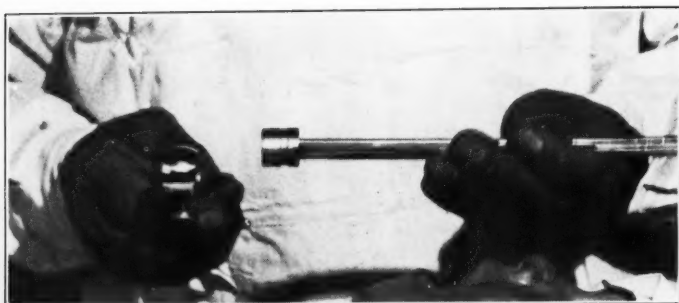


Fig. 5. Bronchoscopic tube and handle about to be assembled and held in place with thumb screw.

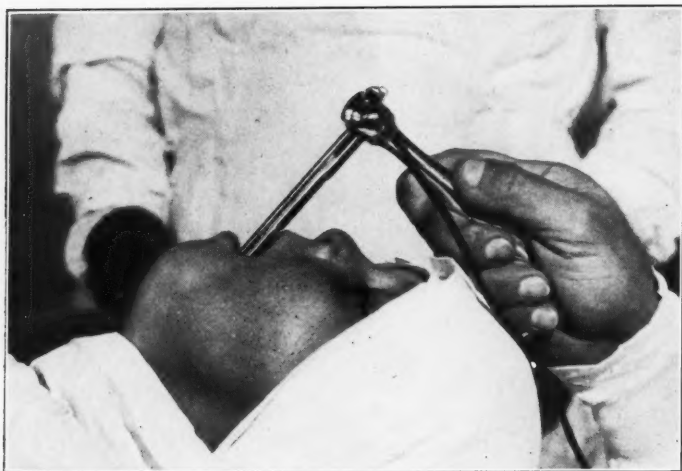


Fig. 6. Instrument in place; introduction complete.
(Any type of forcep, applicator or suction tube can be used through these instruments.)

Advancing by a gentle forward movement of the handle, the tube approaches the vocal cords, which have come into view and are passed, at which time the trachea is entered and the regions beyond are exposed.

No accessory equipment is necessary for this type of direct introduction and all pressure is downward and forward, on the

handle, rather than having the tiresome strain of lifting up, against the tongue and muscle resistance as has been heretofore customary.

After the bronchoscopic tube has passed the vocal cords and into the trachea, the handle may be quickly removed, if desired, permitting the tube to be controlled by the small post of the handle that remains, for the most delicate guidance and manipulation.

Withdrawal can take place quickly and reintroduction is just as easily accomplished as introduction.

In presenting these improved instruments, it is my opinion and the opinion of those who have used them, that bronchoscopy and esophagoscopy have been greatly simplified, especially as it pertains to introduction or reintroduction, in addition to the other features above described.

The cost of the instruments, which is something always to be considered, is identical with the cost of previous instruments of this type. The instruments are made of rustless metal and will not chip or peel.

I desire to express my appreciation to Mr. Thorvald Maijgren, president, and the staff of the Electro-Surgical Instrument Company of Rochester, New York, the manufacturers of these instruments, for their kind co-operation and brilliant results accomplished in bringing this equipment to its present stage of perfection. Their co-operation has played a large part in their successful development.

Since the development of these instruments they have been used under all conditions, on all types of patients, of all ages, without anesthesia, with local anesthesia and under general anesthesia, with an ease and satisfaction hitherto unattainable with any other type of equipment previously available.

Advances in medicine have taken place with a vision into the future, and from necessity, which is the mother of invention.

There can be no progress in standing still.

2010 NIELS ESPERSON BUILDING.

XIV.

THE INFLUENCE OF THE SYMPATHETIC NERVOUS SYSTEM ON THE INTERNAL EAR.*

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The effect of variations in vascularity on the functions of the ear has been a point of discussion for many years. Those conditions which produce a change through an instability of the neurovascular system were studied first, and this led to the experimental production of changes with vasodynamic drugs. Then the changes in vascularity produced by stimulation and interruption of the sympathetic nervous system naturally led to the determination of changes in the middle and external ear and, later, to the effect on the threshold of stimulation of the static labyrinth in animals.

Demetriades¹ states that the earliest experiments, dealing with the effect of paralysis of the sympathetic nerve in the neck, date back several decades. They dealt with the effect produced on the external and middle ear and were reported by Bernard, Duval, Labord and others. Lannois and Gaillard studied the effect of paralysis of the cervical sympathetic nerve on the caloric excitability of the vestibular apparatus in rabbits. They determined an extended period of latency and a shortening of the duration of nystagmus which immediately after intervention was slight, later become more pronounced, and after several weeks returned to normal. Demetriades himself, after severing the cervical sympathetic nerve in guinea pigs, found a complex of symptoms consisting of an increase in tone in the extremity of the affected side, an increase in the excitability of the animal for tactile and sound stimuli transmitted by bone conduction, and a slight reduction in labyrinthine excitability to caloric minimal stimuli. The latter was most marked in the sixth to the tenth week after intervention.

*Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society.

Terracol² says that the autonomic nervous system has a definite influence on the functions of the inner ear. The experimental production of the nystagmic reflex is conclusive in animals and has been verified in man, either by action on the superior cervical sympathetic or the periarterial sympathetic of the internal carotid artery.

Portman and Despons,³ using drugs, found constantly that there was hyperexcitability of the labyrinth after administration of drugs that stimulate the sympathetic system and hypoexcitability after giving depressors.

Szaz,⁴ using drugs which affect the vasomotor system, found that small doses produce symptoms that were interpreted as distension of the small vessels of the inner ear, while symptoms which indicate contraction result from larger doses. He explained the favorable effect of small doses of atropin and the exacerbations of certain vasomotor affections frequently observed after the injection of larger doses on the basis of the state of contraction of the blood vessels.

Ross and Fish,⁵ after experimenting with six drugs on animals, came to the conclusion that the drugs did not have any specific vasomotor effect on the labyrinth, but that the decreased vestibular response was due to the severe depressive reactions on the animal in general. They were unable to produce a hyperexcitability of the labyrinth using the rotation test to determine the excitability.

Draganesco⁶ reported a transitory vestibular syndrome after novocain injection into the neck. The injection was deep in the cervical region and was assumed to have interfered with the superior cervical sympathetic or its carotid continuation.

Portman and Maisonobe⁷ reported a case in which a patient developed violent vertigo after a surgical operation deep in the cervical region. Tinnitus was absent and the vertigo disappeared without treatment. They thought that the sympathetic fibers to the vertebral artery had been interrupted. In their second case they were able to observe a man who had the right carotid ligatured and on the left a periarterial carotid sympathectomy. The sympathectomy produced an increased circulation which decreased

the labyrinthine reactions to caloric stimulation. The ligature did not affect the labyrinthine reactions.

G. Portman,⁸ who has shown considerable interest in the influence of the sympathetic nervous system on the internal ear, states that, regardless of the method by which the vascularity is altered, it is definitely agreed that vasodilation produces hypoexcitability and vasoconstriction hyperexcitability.

Spiegel⁹ discusses the threefold dependence of the inner ear upon the vegetative nervous system. He calls attention to the dependence upon contraction of the afferent vessels upon permeability of the vascular walls and upon fluctuations of the blood pressure. He feels that it is advisable to be conservative in the matter of drawing conclusions, even though a series of observations does show that the vasomotor disturbances have an effect on the inner ear.

The experimental stage that this work is still in is evidenced by the lack of uniformity in the foregoing reports. This, combined with an opportunity to study the effect of interruption of the cervical sympathetic innervation in the human, prompted me in the following studies.

Using eight rabbits, in four of which the cervical sympathetic nerve was severed in the neck, and in four of which the cervical sympathetic nerve was interrupted by alcohol injection, the findings were similar enough to make it possible to report the series as a group. The sympathetic innervation to the side of the head was considered to be interrupted when there was an increase in temperature in the external auditory canal and the homolateral pupil failed to dilate after the instillation of cocain. Kobrak's minimal caloric stimulation method was used to determine the excitability of the labyrinth. All of the animals were tested within twenty-four hours after the interruption, and while there were variations they were not consistent, some showing hypoexcitability and some hyperexcitability. However, in no instance did the period of latency vary over six seconds nor the duration of nystagmus vary over ten seconds. Two of the injected animals were observed for two weeks, and the findings were the same as immediately after interruption. One of the operated and one of the injected animals are still under observation after nine weeks,

and while the findings vary, both in the period of latency and the duration of nystagmus, they did not constantly show either hypoexcitability or hyperexcitability on the affected side, and the variations never exceeded what could be considered within normal limits. The only conclusion that can be drawn from these experiments is that a definite hypoexcitability of the labyrinth on the affected side could not be determined in a period of nine weeks after interruption of the cervical sympathetic nerve. It is possible that changes may be found at a later date. The experiments will be continued and reported in the future.

Since the inner ear of man is more sensitive to vascular changes than that of the rabbit, I felt that it would be valuable to determine the threshold of caloric excitability after interruption of the cervical sympathetic in the human. Five cases were studied in which the cervical sympathetic innervation was interrupted by alcohol injection, operative removal or injury. All of the patients were tested by Kobrak's minimal caloric method, using water at 16° centigrade. Spontaneous phenomena were absent in all cases before intervention. A brief résumé of each case follows:

Case 1.—Mr. C. B., age 32, suffering from asthma, had the inferior cervical and the first, second and third thoracic sympathetic ganglia injected with alcohol, followed by temperature increase in the left upper extremity and left side of head. The left pupil was contracted and did not dilate after the instillation of cocain. He was first examined twelve hours after interruption. After minimal caloric stimulation, nystagmus appeared on the left after forty-five seconds, and on the right after forty-eight seconds, and lasted one minute and thirty seconds on the left, and one minute and twenty-one seconds on the right. Vertigo, induced by mass lavage, was similar in time of appearance, duration and intensity on either side.

Case 2.—Mr. J. M., age 60, suffering from angina pectoris, had the inferior cervical and the first, second and third thoracic sympathetic ganglia on the left side injected with alcohol. The caloric test was first done one month after the interruption. After minimal stimulation nystagmus appeared on the left in forty seconds and on the right in thirty-eight seconds, and lasted one minute

and fifty seconds on the left and one minute and forty-five seconds on the right. Vertigo, after mass douching, appeared in one minute and forty seconds on the left and one minute and thirty-five seconds on the right. No difference in duration or intensity was noted.

Case 3.—J. B., age 44, suffering from arthritis, had the right cervical sympathetic nerve resected in the neck one year before observation. He had a definite enophthalmos on the right and a homolateral miosis. Nystagmus appeared after minimal caloric stimulation in forty seconds on either side and lasted one minute and twenty seconds on either side. After mass lavage, vertigo appeared in two minutes and ten seconds on the right and two minutes and fifteen seconds on the left. The intensity and duration were equal on either side. This patient stated that a tinnitus that he had in the right ear disappeared after the interruption.

Case 4.—Miss M. M., age 32, suffering from arthritis, had the first, second and third thoracic sympathetic ganglia on the right removed and the inferior cervical and the first, second and third thoracic ganglia on the left side injected with alcohol. The excitability of the labyrinths was first tested three months after interruption. On the right side the cervical ganglia were preserved, and there was no evidence of vasodilation and no evidence of the eye signs of paralysis of the cervical sympathetic. On the left side all of the signs of interruption of the cervical sympathetic innervation were present. After minimal caloric stimulation of the right labyrinth, nystagmus appeared after fifty seconds and lasted one minute and twenty-five seconds, and appeared on the left after forty-five seconds and lasted one minute and thirty seconds. Mass lavage was not used on this patient.

Case 5.—Mr. E. E., age 45, was examined five years after an injury in the left cervical region which presented evidence of injury to the cervical sympathetic by the presence of miosis and enophthalmos on the affected side. He also had an area of hyperhidrosis over the left side of the face which could be stimulated by mastication. Although the definite mechanism of production of this phenomenon has not been explained, it is usually considered to be related to paralysis or irritation of the cervical sympa-

thetic nerve. Nystagmus appeared after fifty seconds on either side, after minimal caloric stimulation and continued for one minute and forty seconds on the right and for one minute and thirty seconds on the left. After douching, no difference in time of appearance or duration of vertigo could be determined.

Personal observation of the first two cases and the history of the other three indicated that there was no vertigo, tinnitus or falling at the time of interruption. The general vasomotor signs evidenced by sweating and pallor were the same in all cases, regardless of the side from which they were induced. There was a tendency toward spontaneous past pointing in the extremity on the side of the interruption, in cases one and two, but deviation and past pointing after mass lavage was normal in all cases. Cases one, two and three were re-examined from one to four times in the two months following their first examination. There was no increase in the degree of variation in the threshold of stimulation, indicated by minimal caloric stimulation or mass lavage. The slight degrees of hypoexcitability and hyperexcitability reversed from time to time in some of the cases.

It should be explained that in the cases injected with alcohol for relief of conditions under control of the thoracic sympathetic ganglia, that the inferior cervical ganglion was not purposely injected. Flothow¹⁰ explains that it is almost impossible to avoid the inferior cervical ganglion during the injection of the first thoracic ganglion. However, this procedure of interrupting the inferior cervical and the first and second thoracic ganglia made an ideal setting for experiments that had to do with interruption of the sympathetic innervation to the head because a few strands go up from the upper thoracic ganglia to the middle and superior cervical ganglia, and there might be a possibility that a few fibers were not interrupted if the cervical sympathetic nerve were simply severed in the neck.

It is evident in these experiments on the human, as in the animals, that while there were variations in the period of latency and in the duration of nystagmus, that they did not constantly show evidence of increased or decreased excitability to caloric stimulation. The variation in past pointing can be accounted for

by the increase in muscular tone in the upper extremity of the side of the interruption, which has been found by other investigators.

In the light of the foregoing observations, there are several possible explanations of the absence of hypoexcitability of the labyrinth after the increased vascularity which should be induced by interruption of the cervical sympathetic innervation. From an anatomic standpoint, one theory is that the internal auditory artery, being a branch of the basilar, is dependent for its degree of fullness on the network of nerves surrounding the vertebral artery and its branches and arising from the thoracic strand of the sympathetic nerve, or the inferior cervical ganglion. However, the parasympathetic innervation has not been determined by experiment or histologic examination. While it is usually assumed that such fibers run with the sympathetic, it should be kept in mind that there are old investigations on record that make it seem possible that some of the vasomotor nerves of the inner ear are represented by branches of the cranial autonomic system which find their way into the labyrinth with the auditory and facial nerves and the internal auditory artery. So that it is possible that vasodilation is absent in the inner ear after interruption of the cervical sympathetic nerve.

Secondly, it has been shown on animals that changes are more apparent at a certain length of time after interruption, although I was unable to reproduce it on rabbits after nine weeks. My cases in the human were examined during periods varying from a few days to five years after, but it is barely possible that the period of greatest change was not encountered.

Further, the variations in susceptibility of individuals to changes in vascularity would have to be considered. The group of people having unstable neurovascular systems are more sensitive to influences of the autonomic nervous system than others.

Lastly, the question of unilateral control is mentioned, but Unterberger and de Crinis¹¹ have found that there is a typical reaction in the vasomotor system, regardless of the side from which it is induced.

I do not consider my research exhaustive enough to claim that the working hypothesis of increased vascularity always producing

hypoexcitability of the labyrinth to be incorrect, but that we should be conservative in our deductions concerning the influence of the sympathetic nervous system on the internal ear and that further experimental work and careful clinical observations should be reported.

CONCLUSIONS.

1. The effect on the labyrinth of interruption of the cervical sympathetic innervation in animals and in the human was studied. The first, second and third thoracic ganglia as well were interrupted in some of the cases.
2. Spontaneous phenomena relative to the labyrinth were absent.
3. Variations beyond normal limits in the threshold of excitability of the labyrinth could not be determined.
4. General vasomotor changes, evidenced by pallor and sweating, were the same, regardless of the side from which they were induced.
5. The variations in past pointing can be accounted for by the increased muscular tone in the extremity rather than by labyrinthine influence.
6. The vasodilatory innervation to the inner ear may be part of the cranio-autonomic system.
7. We should be conservative in our deductions on the influence of the sympathetic nervous system on the inner ear and further experimental and careful clinical observations should be reported.

QUIGLEY CLINIC.

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XV.

MICROSCOPIC OBSERVATIONS OF THE PETROUS APEX.*

H. N. GLICK, M. D.,

ST. LOUIS.

Serial sections of temporal bones removed at autopsy were carefully studied. These had been prepared in the Otologic Laboratory, according to the routine celloidin technic, and cut in horizontal and vertical planes. A comparison of conditions within the petrous apex was made with those found in the antrum and mastoid cells in regard to the following points:

1. Degree of pneumatization.
2. Type of marrow.
3. Amount of marrow present.
4. Presence of suppuration in the two regions.
5. Existence of bony dehiscences directly under the Gasserian ganglion.
6. Type of bone found in adjacent sphenoid when present in the section.

Degree of Pneumatization.—In the growing apex various types of bone structure may be present, depending in each case upon the age and other factors influencing its development. The histologic structure in each variety is, however, nearly always determined by its connective tissue matrix. At an early age it generally consists of a delicate ramification of bars and lamellæ of young bone, with no definite tendency towards division into distinct cellular spaces.

In the apex of the two months infant no pneumatization has occurred. The region is filled with marrow, through which ramify delicate trabeculæ of bone. The latter appear as if they were

*Presented at the Oscar Johnson Institute, before the meeting of the Research Conference held April 13, 1932.

From the Department of Otolaryngology, Washington University Medical School, St. Louis.

extensions of bone growing into or surrounded by a solid mass of marrow. The antrum is present but the accessory cells are scarcely pneumatized, as in No. 4330, Fig. 1, and No. 3949; also Fig. 2, No. 3967.

As the process of development advances, however, the trabeculae assume the arrangement of the adult spongy or diploetic bony framework. This consists of denser cross bars and larger spaces. In a five-year-old child (No. 3951), moderately large air cells were observed in the apex on the right side. These occurred

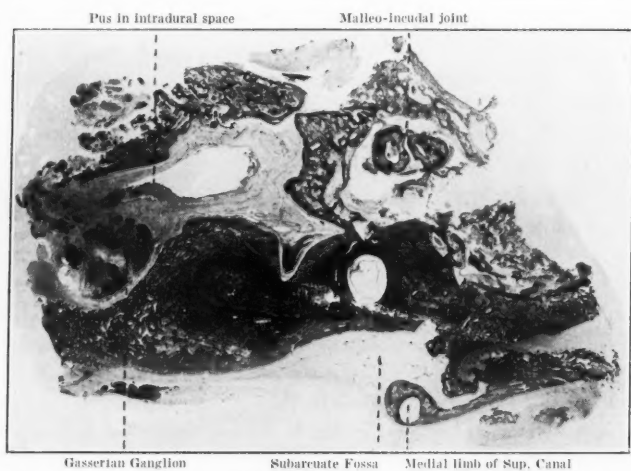


Fig. 1. (No. 4330.) Sec. 200, right, white male, 2 mos. Mastoiditis with pus in intradural spaces near the Gasserian ganglion.

anterior to a region well filled with marrow, as in Fig. 3. Evidently this individual was already developing a pneumatic apex at this age. The left side, however, showed no pneumatization. In a seven-year-old case (No. 3992), however, no air cells were observed, but the region was entirely filled with marrow. One is led to infer that the latter individual was developing a diploetic type.

By 12 or 13 years, the differentiation is evidently established. Specimen 3948, a 13-year-old negro, demonstrates a well pneu-

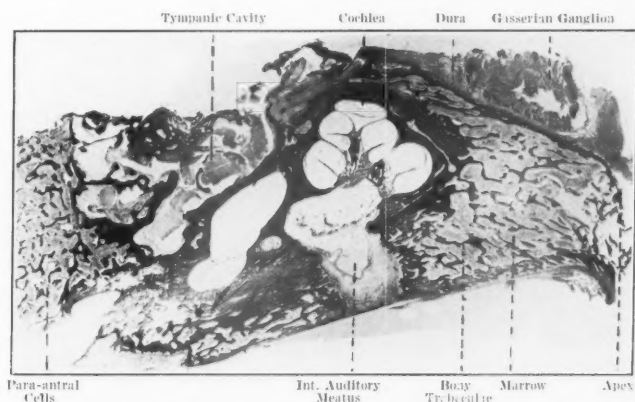


Fig. 2. (No. 3967.) Sec. 470, left, white male, 2 mos. Showing the mastoid and petrous apex regions filled with marrow. The trabeculae appear as if they were extensions of bone growing into or surrounded by a solid mass of marrow.

matized apex on both sides (Fig. 4). In this specimen relatively little marrow occurred in the petrous apex. No adult was found demonstrating such extreme pneumatic development in the apex. The apical cells were larger than those of the para-antral region. As may be seen in the figure, the bony cortex below the ganglion is very thin.

In thirty of the forty-two preparations studied, the pneumatic cells were entirely absent. The latter observation was chiefly found in the growing apex. This was observed in eight infants, ranging from two months to one year of age, and also in three children, 7, 10 and 12 years of age. One preparation of a five-year-old child (No. 3951) showed the absence of pneumatic cells on the left side, but it was found well developed on the right side. The remaining eighteen preparations were in adults from 28 to 62 years of age.

Several microscopic preparations of older specimens revealed a variable number of pneumatic or air containing spaces present in the apex. In the adult the petrous apex may or may not contain marrow. The spaces intervening between the bony septa show considerable variation in their size and number, depending in each instance upon the character of their contents, whether air filled

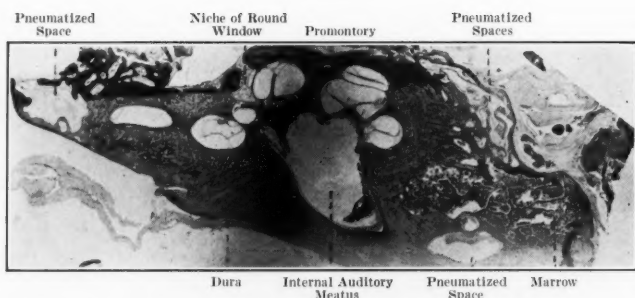


Fig. 3. (No. 3903.) Sec. 347, left, white male, 8 yrs. Fractured skull. Shows thin cortex below which lie pneumatic cells and marrow spaces.

(pneumatic) cells or those containing marrow. As a rule, the right and left sides correspond in type. Nine of the sixteen adult preparations studied contained marrow filled spaces and no pneumatic cells. In these cases the antra were all well pneumatized. In the remaining seven cases, pneumatic spaces were found in the petrous apex. These specimens also possessed well pneumatized antra. As noted above, no adult showed pneumatic cells unaccompanied by marrow containing spaces.

The pneumatic cells are lined by a thin mucoperiosteum, which consists of a tunica propria in close contact with the bony walls, and an overlying layer of flat, nonciliated surface epithelium. Immediately beneath the anterior superior surface of the apex, in the region that lies below the level of the Gasserian ganglion, exceptionally large cells were occasionally found. Some of these measured approximately 18 mm. long by 16 mm. wide. The tegmen in this region, as described elsewhere, may be very thin. Hence, an inflammatory process invading these cells can give rise to pain from direct irritation of the ganglion. An explanation is hereby offered for the pain in the Gradenigo syndrome, as well as for any other sensory disturbances of the trigeminal, that occurs in some cases of acute suppurative otitis media,

Type and Amount of Marrow Present.—One can easily distinguish a variation in the type and amount of marrow that fill the cells in some or all of the areas of the petrous apex. In different preparations there may be found normally a primitive or

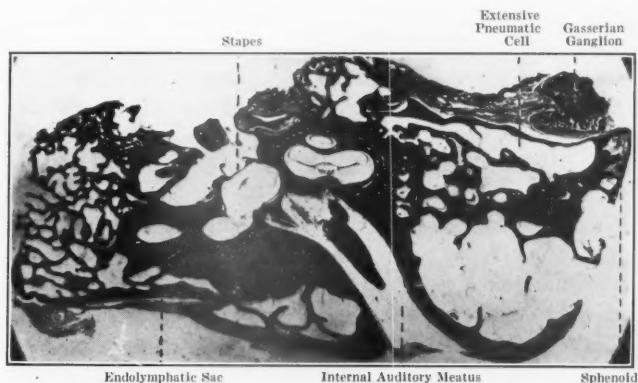


Fig. 4. (No. 3948.) Sec. 681, right, colored male, 13 yrs. Showing large pneumatic spaces just below the Gasserian ganglion. This individual is an excellent example of a purely pneumatic anatomical type.

embryonic type of marrow, also a red or lymphoid and a fatty or yellow marrow. The marrow containing spaces vary in size and number, and appear to have the same intercommunications as the pneumatic cells. When they occur with the air containing cells they are, according to McMahon,¹ closed off from the latter by the tunica propria of the pneumatic cavities, which extend only to the openings of the marrow filled spaces. As a consequence of this arrangement, he says a close communication between the cells is evidently established by the capillaries which are seen to pass between them.

The marrow spaces are essentially filled with blood forming cells of the leucocytic and the hemoglobin holding or erythroblastic series. They also contain within the marrow elements giant cells of the mono- and multinucleated variety (megakaryocytes and polykaryocytes), connective tissue cells, fat cells and endothelial cells. For the sake of clearness the type and amount of marrow found in the spaces are described and tabulated as fatty, intermediate and hyperplastic.

In fifteen preparations the marrow spaces of the apex contained mostly fatty marrow. Pneumatization was not present in these sections and all cells were filled with fat. In some areas of other

sections the cells presented an intermediate type of marrow, the contents being fatty and hyperplastic. In twenty-one preparations studied, however, hyperplastic marrow was chiefly found. This variety appeared to be concentrated in some sections next to the Gasserian ganglion, as in Nos. 3894 and 3942. (See table.)

Presence of Suppuration in the Two Regions.—It has been known for a long time that an intercommunication exists between the system of cellular spaces of the mastoid process and petrous apex. With this fact in mind, therefore, it is not difficult to understand how a suppurative process occurring in the mastoid antrum may extend to the neighboring cells and result in a variable degree of pathologic changes. The character and extent of these changes, however, are in the majority of instances dependent upon the severity of the infecting organism, and particularly upon the type of anatomic cell structure involved. The pneumatic type of mastoid process has been found by others to be more frequently affected by an infection in the antrum than the diploetic, because the marrow containing cells are generally more resistant to an inflammatory invasion than the air cells. This also has been shown to hold for the pyramidal cells.

In a number of cellular spaces anterior to the para-antral region and in proximity to an inflammatory area when present, less proliferative activity was observed in the marrow than in the lining membrane of the air cells. As the two types of cells were followed in some sections anteriorly toward the apex, they seemed to lose the effects of inflammatory invasion, the greater their distance from the antral cavity. In several serial sections examined, the petrous tip cells appeared to show little or no involvement as compared with the purulent reaction when present in the mastoid region. This was especially evident where the process of pneumatization in the petrous pyramid was either poorly developed or entirely absent. Such findings, therefore, lead us to believe that an infection may extend to the petrous apex more readily in those cases in which the cellular structure of the *pars petrosa* possesses a more extensive pneumatic development. This fact also demonstrates clinically the importance and necessity of exenterating completely all diseased pneumatic cells in the mastoid, in order to provide adequate drainage and prevent the inflam-

matory process from advancing into deeper cells. However, as Eagleton has stated,² the bone marrow may become involved with more disastrous results, as some of the microscopic sections show. The laboratory possesses one specimen (four years) in which no mastoid infection may be observed, but a severe osteomyelitis¹ is present in the apex with dehiscence of the cortex.

The presence of pus in the para-antral cells does not always imply that a similar process is to be expected at the same time in the cellular spaces of the apex. While it does occur in certain cases, as shown in some sections, fortunately the suppurative process is generally confined to the mastoid area. In the latter region the character and extent of the pathologic process varied considerably in the different cells. The changes were generally confined to the mucoperiosteum. In some cases the trabeculae were undergoing necrosis and absorption. While some spaces in this region showed signs of early necrosis or were completely filled with pus and hyperactive bone marrow, adjacent cellular cavities presented only a variable increase in vascularity or hyperplastic changes in the lining mucoperiosteum.

In many of the petrous tip cells, however, evidence of increased vascularity was present. An excess of polymorphonuclear leucocytes was seen in the mucoperiosteum of the pneumatic cavities and also in the marrow containing spaces. From the latter also a proliferation of red and white cells seemed to invade the connective tissue spaces of adjacent cavities. Here and there a collection of leucocytic and red cells, probably the result of diapedesis, was seen outside the thin capillary walls. It is through the latter vessels that an intimate anastomosis is said to exist between the pneumatic and marrow containing spaces. However, direct communication frequently exists between the marrow containing and pneumatized cells, just as in the mastoid region. In Fig. 5, No. 3997, Sect. 260 and 300, quite a number of petrous cells were found to be more or less completely filled with serous exudate and in No. 3884 mucopurulent discharge was seen.

While the sequence of an inflammatory invasion in the cells of the petrous apex is usually only that of mild congestion, this has been known, nevertheless, to have resulted in serious complications. Such congestive changes in the cells have yielded, in

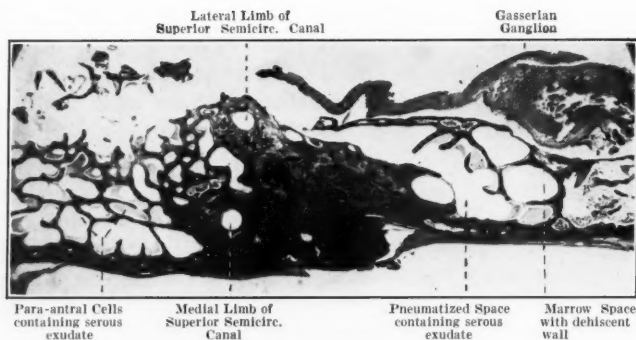


Fig. 5. (No. 3997.) Sec. 260, left, 58 yrs. Showing thin cortex below which lie pneumatized cells and marrow spaces in juxtaposition with the Gasserian ganglion. A small marrow space opens directly under the dura.

some instances, sufficient irritation to cause pain from the trigeminal area or paralysis of the sixth nerve from edematous swelling in the vicinity of Dorello's canal or intracranial complications from direct involvement of the dural sheath covering the petrous pyramid. Fortunately, with the improvement of the middle ear or mastoid suppuration, the pathologic process in the cellular spaces of the apex generally subsides. If the apical infection advances, however, in spite of adequate drainage from the mastoid area, then the process has undoubtedly progressed into the stage of suppuration and necrosis. For the latter condition, therefore, prompt and more thorough surgical procedures have been advocated in an effort to attain recovery.

EXISTENCE OF BONY DEHISCENCES DIRECTLY UNDER THE GASSERIAN GANGLION.

In a number of specimens, the writer has observed the existence of bony dehiscences directly under the Gasserian ganglion (Fig. 6, No. 3917 left, Sect. 171, and No. 3997, Sect. 260). The osseous lamina forming the anterior superior surface of the petrous apex is exceedingly thin in some areas and occasionally, therefore, seems to be a point of predilection for the occurrence of such deficiencies. It is also very evident from this location that the

presence of a dehiscence opening below the dura under the ganglion is in juxtaposition with the trigeminal area and the underlying cells. Under certain pathologic conditions, therefore, a dehiscence may more easily act as a favorable medium for the extension of an inflammatory process to the dural covering with subsequent involvement of the nerve trunks or the ganglion directly.

In some sections, however, where dehiscences were not seen, the underlying cortex was found quite thin. Under these circumstances pneumatized and marrow spaces were lying in intimate

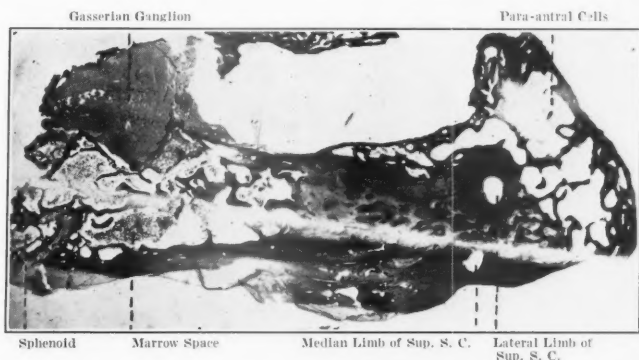


Fig. 6. (No. 3917.) Sec. 135. The section shows what appears to be an invasion of neural tissue into the marrow spaces. Brain tumor-endothelioma. (Unfortunately this section is mounted as if it were one from the right side.) Bony dehiscence under the Gasserian ganglion.

relationship with the ganglion, as in No. 3903, Sect. 347 (fractured skull). In Fig. 6, No. 3917, the section shows a thin cortex and also what appears to be an invasion of neural tissue in the marrow spaces (brain tumor—endothelioma).

No evidence of necrosis was observed about the periphery of the dehiscient areas. In each case the edges of the bone appeared perfectly normal. Such findings were also previously reported by Wolff.³ In her recent microscopic observations of the eustachian tube she concludes that "apertures may occur in the bony wall of the carotid and facial canals, without necessarily having an

extension of the pathologic changes through these interstices." In thin osseous lamellæ it is also possible that dehiscences represent the shallow excavations known as Howship's lacunæ, around which osteoclasts are present on the surface of the bone and have caused the characteristic dissolution of osseous structure from one or more places in the walls. Under such circumstances, therefore, the dehiscence can perhaps be rightfully termed an "osteole," since it represents a vacancy or empty space in the bone, around which no pathology is present.

Defective formation in the osseous shelf below the ganglion was observed more frequently toward the middle fossa than toward the posterior. Occasionally a dehiscence was seen in the roof over the carotid artery. At the distal extremity of the apex and along the posterior border where the cortex is fairly thick, dehiscences were not seen. However, in specimen No. 4078, a dehiscence was found not under the ganglion but along the sphenoid suture.

TYPE OF BONE FOUND IN ADJACENT SPHENOID WHEN PRESENT
IN THE SECTION.

In some sections, as in No. 3884 right, the distal extremity of the apex near the posterior inferior border, appears fused with the sphenoid bone. This may also take place at the articulation with the basilar process of the occipital bone. At times this union is completely ossified in older individuals, and undoubtedly plays a part in preventing a suppurative process from advancing into the adjacent bone.

In six preparations the sphenoid bone revealed numerous fine cells which were filled with marrow. Sections Nos. 3947, 3951 and 4078 were present with hyperplastic marrow, while in No. 4056 and Fig. 6, No. 3917, the sphenoid contained mostly fatty marrow. The occurrence of bone marrow in the sphenoid, usually varies in its amount and distribution, depending in each case upon the stage of osseous development. The sphenoid bone, like the petrous apex, originates from cartilage. This bone in some sections of younger individuals, or occasionally in older children, as in specimen No. 3956, may show cartilage. Specimen No. 4098 shows ossified cartilage and marrow. In specimen 4336, in which

the cells are filled with marrow, a dehiscence in the cortex was also observed.

SUMMARY.

1. A series of microscopic preparations of the temporal bone are described and tabulated with a comparison of conditions found in the petrous apex and mastoid region.
2. Pneumatic cells are present in the apex of the human temporal bone more frequently than is generally believed.
3. The possibility of a dehiscence occurring in any bony wall must be constantly kept in mind.

3115 S. GRAND BLVD.

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TABLE I (A).

Autopsy Number	Side	Cut	Age	Race	Sex	Splenoid	Marrow		
							Fatty	Intermediate	Hyperplastic
3967	L	H	2 mos.	W	M	Not seen			+
3967	R	V	2 mos.	W	M	Not seen			+
4330	R	H	2 mos.	W	M	Not seen			+
4330	L	H	2 mos.	W	M	Not seen			+
3949	L	V	2½ mos.	W	F	Not seen			+
3949	R	V	2½ mos.	W	F	Not seen			+
3865*	L	H	4 mos.	W	M	Not seen			+
4078	R	H	5 mos.	W	M	Present with fatty marrow Not seen			+
4078	L	H	5 mos.	W	M				+
3920	L	V	6 mos.	W	F	Not seen			+
3920	R	V	6 mos.	W	F	Not seen			+
3947*	L	H	7 mos.	C	F	Appears with hyper-plastic marrow			+
4106	R	V	1 yr.	C	F	Not seen			+
4106	L	V	1 yr.	C	F	Not seen			+
4048*	L	H	2 yrs.	W	F	Not seen			+
3951	R	H	5 yrs.	W	F	Appears with hyper-plastic marrow Not seen			+
3951	L	H	5 yrs.	W	F				+

TABLE I (A)—(Continued).

Autopsy Number	Apical Pneumatization	Dehiscence	Diagnosis	Mastoid Condition
3967	None	Not seen	Congenital hydrocephalus.	Not pneumatized, except the antrum.
3967	None	Yes		
4330	None	Not seen	Clinical stricture of esophagus.	Accessory antral cells scarcely
4330	None	Not seen	Otitis media, Mastoiditis.	pneumatized. Full of marrow. Heavy cortex —not pneumatized.
3949	None	Not seen	Anytoma congenita.	Heavy cortex — not pneuma-
3949	None	Not seen		tized.
3865*	None	Not seen	Otitis media, right and left. Bronchial pneumonia.	Pus—not pneumatized.
4078	None— Narrow	Not under ganglion but along splenoid suture.	Influenzal meningitis.	Main antrum fairly large. No accessory cells.
4078	Narrow	Not seen		Blood forming marrow.
3920	None	Not seen	Hydrocephalus acquired.	Antrum cavitated but acces-
3920	None	Not seen		sory cells full of hyperplastic marrow. Pus, particularly in right.
3947*	None— Infection	Not seen	Influenzal meningitis.	Thick cortex. Marrow—pus.
4106	None	Not seen	Tuberculosis—meningitis.	Pus. Mostly filled with marrow in accessory cells.
4106	None	Not seen		Blood forming marrow.
4048*	Toward the middle fossa	Not seen	Influenzal meningitis.	Pus. Well pneumatized.
3951	Yes	Not seen	Meningitis—otitis.	Well pneumatized.
3951	None	Not seen		Pneumatized.

TABLE I (B).

Autopsy Number	Site	Cut	Age	Race	Sex	Spleen	Marrow		
							Fatty	Intermediate	Hyperplastic
3992*	R	H	7 yrs.	W	F	Not seen	+	to	+
3993	R	H	8 yrs.	W	M	Not seen		+	to
3993	L	H	8 yrs.	W	M	Not seen		+	to
3956**	R	H	10 yrs.	W	F	Cartilage		+	to
3956	L	H	10 yrs.	W	F	Not seen		+	to
4336	R	H	12 yrs.	W	M	Present with marrow. Deliscent. Not seen	+	to	+
4336	L	H	12 yrs.	W	M	Not seen	+	to	+
3948	L	H	13 yrs.	C	M	Appears with marrow		Relative-ly little	
3948	R	H	13 yrs.	C	M	Appears with marrow		Relative-ly little	
4142	R	H	28 yrs.	W	M	Present with marrow	+		
4142	L	H	28 yrs.	W	M	Not seen	+		
4056	L	H	31 yrs.	C	M	Present with fatty marrow	+	Mostly fat	
4056	R	H	31 yrs.	C	M	Not seen		Mostly fat	
3884	L	H	36 yrs.	W	M	Appears fused with petrous	+		
3884	R	H	36 yrs.	W	M	Not seen	+		

TABLE I (B)—(Continued).

Autopsy Number	Apical Pneumatization	Dehiscence	Diagnosis	Mastoid Condition
3992*	None	Not seen	Spinal meningitis.	Well pneumatized. Contained serous fluid.
3903	Evidence of good pneumatization toward middle ear	Not seen	Pachymeningitis, following fracture of skull.	Extreme but with thickened submucosa.
3903	Both marrow and air	Not seen		Pneumatized—Infection.
3955** 3956	None None	Not seen Not seen	Thrombophlebitis — Chronic otitis media with cholesteatoma.	Marrow and air spaces, also infection.
4335	None	None	Mastoiditis—Lateral sinus thrombosis.	Well pneumatized.
4335	Beginning fatty marrow	Heavy cortex		Well pneumatized. Infection.
3948	Extreme. Huge air cells.	None. Cortex quite thin	Multiple bone tumor.	Pneumatized. Small cells.
3948	Extreme	Not seen		
4142	None. Filled with marrow.	Yes	Cerebral glioma astrocytoma, temporal lobe, right.	Pneumatized. Pus.
4142	None. Filled with marrow.	Not seen		Pneumatized.
4056	Filled with fatty marrow.	Not seen	Lucid meningitis.	Well pneumatized. Extremely large cells.
4056	Fatty marrow with few air cells.	Not seen		Well pneumatized.
3884	None	Yes	Multiple areas of cerebral softening, especially white matter. Recent craniotomy.	Well pneumatized; mucopurulent discharge in.
3884	None	Not seen		Pneumatized.

TABLE I (C).

Autopsy Number	Side	Cut	Age	Race	Sex	Spleenoid	Marrow		
							Fatty	Intermediate	Hyperplastic
4005*	L	V	44 YRS.	C	M	Not seen	+		
							Mostly fat		
3897*	L	H	45 YRS.	W	F	Not seen	+		
3917*	L	H	47 YRS.	C	F	Appears with marrow	+		
4098*	R	H	48 YRS.	C	F	Ossified cartilage and marrow	+		
3894	L	H	51 YRS.	W	M	Not seen	Marrow filled space immediately next to ganglion in inferior level.		
3894	R	H	51 YRS.	W	M	Not seen			
3997*	L	H	58 YRS.	W	M	Not seen	+		
3912*	R	H	60 YRS.	W	M	Not seen	In places purely fatty, rather hyperplastic just near ganglion.		
4063*	R	H	62 YRS.	W	M	Not seen			
								+	
								Mostly fat.	
3985*	R	H	62 YRS.	C	M	Not seen	+		

TABLE I (C)—(Continued).

Autopsy Number	Airial Pneumatization	Dehiscence	Diagnosis	Mastoid Condition
4005*	Yes	Not seen	Lues. Cerebral hemorrhage.	Well pneumatized.
3897*	None	Yes	Brain tumor.	Extreme pneumatization.
3917*	None	Yes	Brain tumor.	Pus in mastoid. Well pneumatized. Main antrum appears a bit small. No fat or marrow.
4098*	None. Filled with fat throughout.	Yes	Chronic mastoiditis, left. Diabetes mellitus.	Well pneumatized. Large cells.
3894	Extensive air space next to ganglion at superior level.	Not seen	Spongiblastoma multiforma. Left parietal temporal lobe.	Well pneumatized.
3894	Pneumatic and fatty cells in apex.	Not seen		Well pneumatized.
3997*	Extreme. Huge cells.	Yes	Cancer of face. Neck abscess. History of syphilis.	Extreme.
3942*	Extensive; more on the side toward the middle fossa, than on the lateral.	Of marrow spaces around ganglion, polyps, round cells, etc.	Takes dorsalis syphilitic cerebrospinal meningitis.	Well pneumatized.
4063*	All cells filled with fat.	Very thin cortex	Cerebral hemorrhage and cancer of mouth.	Highly pneumatized.
3985*	Eburnized bone with a few small cells.	Not seen	Chronic pulmonary tuberculosis. Arteriosclerosis and syphilitic enditis. Disseminated tubercles.	Well pneumatized.

*Material unobtainable from the opposite region.

**This specimen was previously described by Birsner and Wolff. *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY*, Dec., 1930, Vol. 39, p. 1045.

XVI.

WAVES VS. VIBRATIONS IN OTOTOLOGY.

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Modern scientists are using the words "waves," "currents," "vibrations" and "radiation" interchangeably. What significance has this for the otologist in the problem of deafness? It is fundamental, for the theory of hearing was built upon the supposition that the atom was the indivisible part of matter and that molecules or particles of matter moving through the ether carried sound impulses by way of the external auditory canal to the organ of Corti. We shall try and show that the electron has taken the place of the atom and electric units of the molecule as a structural basis of matter. It would be correct, then, to use the word "waves" as synonymous with "vibrations" or "radiation." This position cannot be assumed lightly, for we cannot have sound impulses carried by waves that originate from electromagnetic currents and also waves due to particles of matter moving through the ether, for they would be subject to different laws. The answer to this question for the otologist is not wholly academic, for, on the supposition that sound impulses are carried by particles of matter in motion, obstructions in the middle ear would have a marked effect in impairing normal hearing. Sound impulses, however, that were propagated by radiation would not be affected by such obstructions. The whole theory of the origin of sound impulses and the function of the conduction apparatus depends on whether waves can be used to indicate molecular motion or whether such motion is due to vibrations or radiation. In order that we may understand the phenomena of matter in motion, such as light, sound, heat and electricity, we must first determine its structural basis. It is obvious that such a basis must have as its foundation the ultimate division of matter and, since the atom has been split into electrons, such electrons must be our starting point. Now, what are the laws that govern the motion of electrons? As the question is one relat-

ing to physics, we can do no better than to review some of the recent work on that subject.

The mathematicians were very much upset by Einstein's theory of relativity, but much more so by Thomson's and Rutherford's work on the electron, in which it was shown that the atom could be further divided into electrons and protons. Moreover, the electrons and protons followed the law of electric matter whose phenomena are expressed by means of vibrations or radiation. This is indicated by Fitzgerald's law, which is as follows: "The electric theory of matter makes any standard measurement variable because electric units set up electromagnetic currents in matter, which are followed by contraction and later by expansion." Einstein puts it in this way: "There is a variant and relative standard measurement for each planet, for each planet has a different speed of rotation." This variant for short measurements could be ignored, but in measuring the distance of a planet from the earth the variant is of great importance to scientists. Sir James Jeans has explained the foregoing laws very clearly: "If a metal bar of iron or steel is placed on its side, the electric units are in a state of contraction. If, now, the bar is set on end, the electric units expand and the variant is established, as the same bar is now longer." Sir James Jeans goes on to add, "gravitation has been driven out of physics and discontinuity has taken its place."

So far, we have seen that standard measurements are only relative and that we are dealing with electric units in matter.

RADIATION.

The theory of light is that it is due to the radiation of the sun's rays, or, in other words, electronic vibrations. We cannot prove that there are any waves around the sun (Jeans), or, indeed, that there are waves anywhere. We do not know that there is any ether (Eddington). The molecular wave theory has been used by the mathematicians as a working hypothesis. As we are now dealing with electric matter, we must look upon light and sound as being due to vibrations. Vibrations cannot be considered to produce waves in the sense that the term was used by Helmholtz, for they are subject to different laws. If, then, we try to measure

radiation or energy by means of waves we are using mathematical formulæ that do not apply to electric matter. The law of radiation, as laid down by Einstein, is as follows: "Radiation of a given type can affect an atomic or molecular charge only if the energy needed for the charge is precisely equal to that of a single quantum of radiation." We must keep the quantum theory in mind later in connection with Jeans' statement when measuring the radiation of the atom.

ATOMS.

"The atom is bipolar, one positive charge in the proton being equal to two negative charges in the electron. The atom, besides carrying a positive and a negative charge, has electromagnetic energy. The atoms move at terrific speed in their orbits." (Jeans.) "The ninety-two chemical atoms are bipolar and radio-active." (Shapley.) The breaking up of a radio-active atom has been likened by Jeans to the discharge of a gun. While Eddington says, "Following such a discharge there seems to be a period of rest as though the atom was being recharged." Jeans also states that "light waves, however intense, do not affect the motion of the atom unless the number of the waves are just right, whereas the most feeble vibrations act at once if the number of vibrations are right." This is a significant statement when taken in connection with the measuring of the radiation or energy of the atom by the mathematician. Jeans concludes as follows: "When I should logically use the measure method, I know from experience that it will not work, and so I use the sweepstake method, and we do not know why."

CELL.

Crile has shown the living cell to be bipolar and has demonstrated an electric current.

SOUND.

The greatest advance in our understanding of the transmission and reception of sound impulses has been made through the marvelous development of the radio. In one of the latest books on electricity by Ernest Greenwood is the following on electrical

communication without the use of wires: "Radio communication is carried on by invisible electromagnetic waves sometimes called electric waves. Electromagnetic waves are a combination of electric and magnetic phenomena which are set in motion by apparatus and devices that may be broadly covered by the term 'transmitter.' The transmitter consists mainly of apparatus for generating high frequency currents. It also embodies an antenna or aerial, which may consist of one or more elevated wires supported from a tower, pole or other structure. When the antenna or aerial is fed with high frequency alternating currents, electromagnetic waves are automatically radiated into space to all points of the compass. Currents of very high frequency, say of the order of 3,000,000 to 20,000,000 per second, radiate short waves, while currents of frequencies from 3,000,000 down to 15,000 per second radiate what are termed long waves. So long as these currents flow in the aerial system electromagnetic waves travel outward. Start and stop these currents by a telegraph key and one can make the wave motion simulate the dots and dashes of the Morse code. Control them by a microphone or telephone transmitter and one can transmit the acoustical undulation of speech or music. To detect these waves we erect another elevated wire at a distant point—that is, at a receiving station. This wire will absorb some of the energy of the passing wave in the form of feeble alternating currents of the same frequency as those generated in the transmitter. These high frequency currents thus induced into the receiving wire cannot be made to work directly any device that will produce an effect on the human ear. It is, therefore, necessary to transform these currents so that they will operate a telephone receiver or a loud speaker."

From the foregoing detailed description of the practical working of the radio in transmitting and receiving sound impulses, it is clear that the writer assumes that waves are due to electromagnetic currents and that sound impulses are propagated by vibrations or radiation. This assumption would support the statement of Reymond that sound impulses are propagated by electricity. If more evidence was needed, Jeans declares that the charge from one motion to another motion is effected by radiation. Long and short waves, then, are long and short radiations.

Our study further shows that the molecular wave theory of Helmholtz upon which he built up his theory of hearing is no longer tenable, for waves initiated by particles of matter moving through the ether would not conform to the laws of electric matter. We have now traced motion or sound from its inception to its transmission by radiation to the receiving station when it was amplified for its reception by the human ear. We will now consider the auditory receiving station and its equipment for receiving such impulses, according to the law of electric matter. Shapley says that all known chemical atoms are bipolar and radio-active, while Crile has demonstrated that the human cell is bipolar and has electromagnetic energy. The filaments of the auditory nerve end in the magnetic field of the receptor cell. In our review of the atom we remember that Jeans stated its energy or motion was effected by the most feeble vibrations, provided that they were of the right number. Crile adds that the cell, besides having electromagnetic energy, is a self-charging conductor. Having in living tissue the necessary structure for the reception of sound impulse propagated by radiation, we can understand that the relay of cells might act as transformers in stepping up or down weak impulses in the same manner that weak currents are stepped up and amplified for reception by the human ear at the radio receiving station. The splitting of the atom has brought into play new forces that are of vital importance to the otologist in his conception of waves and sound conduction in approaching the problem of deafness. This is shown by the fact that granulations and scar tissue are no obstruction to sound impulses propagated by radiation. In other words, mechanical or physical causes are not operative in the loss of tone perception in slowly progressive deafness. The conduction apparatus, having no physical function to perform in normal hearing, becomes a protective mechanism only. This would explain why the Politzer bag and bougie has failed to help any case of chronic deafness. In 1920, the writer published in the *New England Journal of Medicine* an article entitled "The Minor Rôle of the Conduction Apparatus in Slowly Progressive Deafness," in which he stated that in every case of so-called middle ear deafness there was a nerve element. In several articles published in the *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY*

it was claimed that when there was a loss in tone perception, together with tissue changes in the eustachian tube and middle ear, following a focal infection, the pathology in the tube and tympanum were coincident and not a factor in the resulting deafness. One was a physical and the other a nerve impairment, the damage to the auditory nerve mechanisms being caused by a bacteremia through the blood stream. Hearing tests have been published repeatedly showing a gain of 10 to 50 per cent in tone perception following the removal of a focal process in adults without any direct treatment of the middle ear, only to have such gains lost by subsequent reinfection. Clinical experience and physics then unite to rule out the conduction apparatus as a factor in chronic deafness. For the past 100 years we have based our understanding of matter in motion on a theory. From a study of modern physics and the structure of living tissue we now find that motion conforms to the general laws of electric matter. Crile reached the same conclusion from his laboratory work, the steps of which he has embodied in his "Bipolar Theory of Living Processes." If deafness is due to impaired nerve function then we must remove the cause, desensitize the patient to that type of infection and build up his resistance to prevent reinfection. Having arrested the intermittent progress of the deafness, the problem ahead of us is to see if we can restore any of the lost function of the auditory nerve.

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XVII.

ANTROSCOPY AND ITS RELATION TO THE ANATOMY OF THE MAXILLARY SINUS.

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The diagnosis of maxillary sinusitis is dependent upon history, transillumination, antral washing, and Roentgenograph, with or without radiopaque media. During the last two decades there has been developed a new procedure in this diagnosis, called antroscopy or antroscopic examination of the maxillary sinus. This procedure seeks to make the diagnosis by direct vision of the lining membrane, which would seem to be a highly desirable method of diagnosis. Several men have written up this procedure, notably Baum,¹ who punctures through the canine fossa into the maxillary sinus. Haseltine² and Maltz³ also preferred puncture through this wall of the sinus, while Spielberg^{4 5} punctures through the inferior meatus in the nasal fossa. There have been some variations in the type of instrument used, but essentially the instrument is the average nasopharyngoscope. The success of diagnosis by this means gives variable results and has not come up to probable expectations. As to the comparative value of the nasopharyngoscopic diagnosis of antrum disease against our present methods, I feel certain that the advantages are markedly and distinctly toward seeing the pathology and even possibly palpating it with a probe, if possible, as against our present indirect methods of X-ray, washing and transillumination. This comparison has already been presented,⁶ and it was with this in mind that I called attention to our limited use of the nasopharyngoscope as a means of direct certain diagnosis of disease in the maxillary sinus. At that time mention was made that only puncture through the inferior meatus had been used, not the route through the canine fossa. Following the appearance of this article I received a letter from Dr. Burton Haseltine of Chicago advising me to try

the last mentioned route since "it has many advantages over the use of the scope through the nasal chamber."

With the purpose of determining the advantages and objections of each of these routes and the general results of antroscopy, the following work was begun to determine the problems as follows:

1. To what distance could the lens of the antroscope be moved from its entry (a) through the inferior meatus and passed posteriorly and laterally (b) through the canine fossa and passed superiorly? The conclusion to be drawn from this was: the greater the distance the lens could pass from its point of entry the more certain of seeing more of the lining and its pathology.

2. Which route permitted seeing the ostium maxillare more frequently and more fully? It was this last factor that soon brought my attention to the next problem.

3. What is the length (mediolateral dimension) of the so-called ostium of the maxillary sinus (not its dimensions in the sagittal plane); or is this opening really a canal or duct?

The procedure for the first problem was as follows: To puncture the inferior meatus with the cannula-trocar combination furnished with the antroscope. Then remove the trocar and insert the antroscope. If the lens of the antroscope could see into the antrum, as it usually could, notations were made as to how much of the antrum could be inspected, and as to how much pathology could be seen. Then the cannula was moved to just cover the most anterior fragment or piece of bone broken off from the puncture through the inferior meatus as seen within the antrum with the antroscope. The cannula was held fixed at this point and the distance that the antroscope could be moved posteriorly and laterally until it hit the posterior or temporal wall was measured. This is clearly visualized in the drawing labeled Figure 1. The same examination through the canine fossa was then made and a similar procedure carried out. The cannula being fixed at the fragment of bone fractured at the inferior margin of the puncture in the canine fossa as viewed within the antrum, the antroscope was moved superiorly, and the distance traversed, until it was stopped by the roof, was measured.

The procedure for the second problem was to note if and how much of the ostium of the antrum could be seen with the antro-

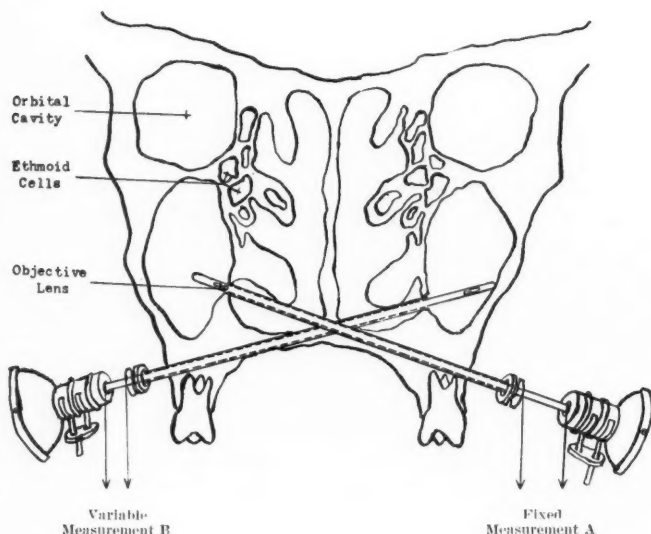


Fig. 1. Drawing to show how the distance that the objective lens moved into the antrum was obtained; measurement *B* was subtracted from that of *A* (of course both measurements were taken on the same antrum).

scope passed through the inferior meatus and similarly when it passed through the canine fossa. Finally, if the ostium could not be seen, a probe would be passed to determine whether the ostium had been in the field of vision and not recognized. This would happen usually in the "slit" type of opening into the antrum.

The third procedure was to remove any one of the walls (not the base) of the antrum if it had not already been done. Then the ostium or ostia were probed with a graduated pliable probe. Several Yankauer eustachian bougies, graduated in 2 mm. markings and unusually resilient, served well for this purpose. Then the distance from the border of the hiatus semilunaris—i. e., from the sagittal plane in which the uncinate process lay to the antral border of the ostium of the maxillary sinus—was measured. Following this, another measurement was made, viz., from the floor of the infundibulum—i. e., its lateral wall to the margin of the ostium in the maxillary sinus. This last measurement was to

determine the depth or length of what is called and known as the ostium maxillaris.

With all this data completed and the antrum lining exposed the mucosa was inspected and an actual check on the antroscopic observation recorded. Occasionally there were definite errors in the antroscopic observation either of omission or commission, as can be seen in Table I.

PROBLEM NO. I.

To come back to the first problem of whether the meatal or canine route permitted the lens of the antroscope to pass deeper into the sinus, with the view of examining more of its lining: There were thirty-four antra used for this determination, two of which were female. Undoubtedly, there were more female crania, but these were not definite enough to list as such. In puncturing through the inferior meatus, the number of millimeters that the lens of the antroscope could pass the margin of entry varied from the extremes of 11 mm. in specimen 9R. to the low of —3 mm. in 7L.—i. e., the objective lens was short 3 mm. of being permitted into the antrum. Naturally nothing could be seen of the lining of the antrum with the antroscope puncturing through the inferior meatus in this specimen. Of the thirty-four antra there were in all three such specimens, namely, 7R., 7L., and 12R. The average number of millimeters that the objective lens could pass through the inferior meatal puncture for seventeen right-sided specimens was 4.85 mm., if one deducts the 1 mm. for specimen 7R., and 4.26 mm. for seventeen left-sided antra, also deducting 3 mm. for 7L.

The canine fossa route permitted the lens to enter the antrum as high as 24 mm. in 3R., and as little as 0.5 mm. in 5L. The average for the seventeen right-sided specimens with a puncture through the canine fossa was 13.41 mm., and for the seventeen left was 12.46 mm. The average for thirty-four specimens through the inferior meatal route was 4.56 mm. against 13 mm. for the canine route, practically 4.5 to 13 mm.

J. Parsons Schaeffer⁹ gives the average inferior superior dimension of 150 antra as 33 mm. In these thirty-four antra the average length the objective lens could move from the canine fossa to the

TABLE I.

Specimen Number	Length Canine Entry in mm.	Observation Through Meatal Entry	Length Canine Entry in mm.	Observation Through Canine Entry	Undulate Ridge to Ostial Margin in mm.	Length Canal in mm.	Remarks
1R.	8.	Ostium almost seen. Fair view of antrum.	11.5	Ostium seen, good view of antrum, as a whole.	10.	6.	Canal is directed ant., inf.
1L.	4.	Ostium not seen; limited view in sinus.	9.5	Ostium seen, fair view of remainder of antrum.	13.	6.	Canal directed inf., lat.
2R.	2.	Ostium not seen and little of mucosal wall.	14.	Ostium seen; fair view of inside of sinus.	10. sup. 7. inf.	8. 5.	*Canal directed inf., lat. There was a polyp on the medial wall not recognized antroscopically. (B)
2L.	2.5	Ostium seen with marked shifting of "scope"; no nasal septum present.	13.5	Ostium seen, and good portion of antrum.	6.	2.	Canal directed inf., lat. Small sessile polyp just ant. to ostium, not recognized with the antroscope.
3R.	7.5	Ostium recognized only after probe was passed.	24.	Ostium not seen; a ridge in antrum obstructs view of it.	13. post. 8. ant.	9.5 4.5	*Canal directed ant., inf. Polyp on base just above meatal puncture (B).
3L.	4.	Ostium not seen; can hardly see into sinus.	17.	Ostium not seen, being near roof. With extreme shifting and view seen in periphery of view.	4.	Canal directed ant., inf. There really is no bulla, it being so markedly compressed by a deflected septum (C).
4R.	6.5	Ostium not seen; portion of antrum seen.	13.	Ostium seen, and fold of mucosa making a sort of wall for a canal.	6.	2.	Canal directed laterally.
4L.	9.	Ostium is seen.	19.	Ostium is seen, small in size.	8. sup. 6. inf.	4. 2.	*Canal directed ant. Ostium lay just below roof of antrum.

5R.	3.5	Ostium just barely seen.	5.	Ostium seen; lacrimal bulge discerned just post. to the nasal puncture.	10.	6.	Canal directed inf. Through canine puncture was seen another directed ostium, really an infraorbital recess.
5L.	5.	Natural ostium barely seen; accessory, not.	0.5	Neither ostium seen; all that can be observed in this view is to see mental puncture.	Natural ostium 13. post. 9. ant. accessory ost.	10. 6. 1.	*Natural canal directed post., inf. Required mallet to puncture canine fossa. (D)
6R.	3.5	Ostium barely seen.	13.	Apparently two adjacent ostia seen.			Specimen lost before final data could be completed.
6L.	2.		14.	Saw some of the mucosal wall.			Medial wall of this specimen destroyed.
7R.	—1.	Obtained a glimpse into antrum by turning lens to view of medial wall of sinus.	11.	Ostium not seen; recognized thickened membrane.	13. post. 10. ant.	10. 6.	*Canal directed inf., post. Membrane was markedly thickened; numerous sessile polyps. Ostium was just under the roof.
7L.	—3.	Cannot see into the antrum.	5.5	Ostium not seen; only small portion of lining seen. Membrane is thickened.	14. post. 12. ant. 4.	10. 8. 3.	*Canal is slit-like; roughly, direction is inf. lat. Second canal is directed lat. Membrane is markedly thickened.
8R.	8.	Ostium is a slit, not recognized until probed.	20.	Ostium recognized only after probing.	6.	2.	Canal is directed inf., lat. Ostium lies sup. and ant. in antrum.
8L.	4.	Ostium not seen; can see but little into antrum.	19.	Ostium seen.	6.	1.	Canal directed ant., inf.
9R.	11.	Ostium seen, appears as two adjacent ostia, separated by a mucosal band.	18.	Two ostia seen.	Normal duplicate ostia a- 9. post. 6. ant. b- 9. post. 6. ant. c- Accessory ostium	4. 1. 5. 7. 1.	*Canal directed post., inf. *Canal directed post., inf. Lies post. to hiatus semilunaris; canal directed lat. (E) c-Canal directed laterally.

TABLE I—(Continued).

Specimen Number	Length Meatal Entry in mm.	Observation Through Meatal Entry	Length Canine Entry in mm.	Observation Through Canine Entry	Uncinate Border to Ostial Margin in mm.	Length Canine In mm.	Remarks
9L.	8.	Ostium seen.	12.5	Two ostia seen.	a-10, post. b-0 ant. c-accessory ostium ant. post.	4. 2. 2. 1. 8. 6.	a-Canal directed inf. and slightly post. b-This canal is completely distinct from (a); it lay at tail end of a flattening hiatus semilunaris and was directed lat. c-Canal is directed post. (E)
10R.	3.	Ostium just seen.	11.5	Ostium barely seen in upper pole of view; appears small	9.	7.	Canal directed post., inf.
10L.	2.5		12.5				Portion of medial wall containing ostium is destroyed.
11R.	6.	Ostium not seen.	8.	Cannot see ostium.	a-10, Duplicate ostia b-8.	6. 4.	a-Canal is separate and distinct from duplicated one below, and is directed ant., inf. b-Canal directed inf., post. Has smaller opening than (c) with common infundibular opening. c-Canal directed post.
11L.	2.5	Cannot see ostium; fragment of bone obstructs view.	5.5	Ostium barely seen.	c-10. 10.	5. 4.	Canal directed inf., lat.
12R.	0	Can just see into sinus when lens is turned to look medially.	10.	Polyps seen, two on medial, one on ant., one on post. wall. Ostium discerned after probing; seen to lie between two polyps.	Normal ostium a-11. b-Accessory opening	8. 10.5	a-Canal directed lat. Membrane is markedly thickened. b-This canal uses the same antral opening as (a), even though the opening is below the uncinate.

13R.	8.5	Ostium can not be seen.	18.	Ostium is discerned.	10. ant. 8. post..	4. 2.	*Canal is directed lat., inf.
14R.	4.5	Only the post. of the two ostia is seen.	15.5	Two ostia seen; post. one might be easily missed.	Ant. ostium a-9. Posterior ostium b-5.	4. 2.	a-Canal directed ant., inf. This is a pair of duplicate ostia. Made puncture canine fossa. b-This canal directed lat.
14L.	1.5	Lower half of ostial margin seen after opening was probed.	11.	Ostium is seen. Thickening of membrane can be discerned, granulation-like tissue.	8.	6.	Canal directed lat., inf. Both the inf. meatus, and the canine fossa required aallet to puncture them.
15R.	2.	Two ostia seen; piece of tissue mass seen on floor.	7.5	Ostium not recognized until probed; must in periphery of view. Foreign tissue seen. Nasal puncture went thru lacrimal bulge.	6.	1.	Canal directed post., inf. Tissue proved to be a piece of coagulated blood. Could not probe post. adjoining ostium, the mucosal band making the double canal seem too close to the inferior floor, through the canine fossa; only the large ant. ostium was seen.
15L.	9.	Ostium a slit, and not recognized until probed. Polyp recognized in roof.	11.	Ostium cannot be gotten into view, only small portion of polyp seen.	8.	3.	Canal directed post. Polyp proved to be a piece of coagulated blood stuck in roof of antrum.
16L.	5.	Ostium not seen; fragment of bone in lower part of floor; piece of probe seen on floor.	12.5	Two ostia seen; can look thru canal of one. Polyp clearly seen, and piece of metal probe.	a-10. b-Accessory opening.	6. 7.	a-Canal directed lat. b-This is an accessory ostium, directed lat. Lies post. to hiatus semilunaris. Polyp and piece of metal probe present.
17L.	3.	Recess into ethmoid region recognized.	17.				Ostium destroyed in this specimen.

TABLE I—(Continued).

Specimen Number	Length Meatal Entry in mm.	Observation Through Meatal Entry	Length Canine Entry in mm.	Observation Through Canine Entry	Uncinate Border to Ostial Margin in mm.	Length Canal in mm.	Remarks
18R.	1.	Ostium cannot be viewed.	9.	Ostium seen. There appeared to be another opening.	9. Inf. 5. sup.	6. 2.	*Canal directed post., inf. Through canine fossa was seen to be what appeared to be an ostium, but it was an infraorbital recess.
18L.	4.5	Ostium just seen; also recess resembling an ostium.	17.	Ostium seen well; also recess space.	7.	5.	Canal directed inf., post.
19L.	9.	Small ostium well seen and recess too.	15.	Ostium as well as remainder of antrum seen.	10. ant. 8. post.	6. 4.	*Canal directed lat., post.
20R.	8.5	Ostium prevented from being seen by fragment of bone.	19.	Ostium well seen; antrum also.	5.	1.	Canal directed inf., post.

*Frequently in the atral margin of the ostium there is a fold of mucous membrane, at times of bone, projecting into the antrum, making the wall of that side of the canal longer in the mediolateral dimension. Naturally the other side is shorter, it being flat and even with the surrounding sinus wall.

(B) The polyp present in this specimen was not recognized because it looked as if a piece of bone and its covering mucosa had been pushed into the sinus.

(C) There really is no infundibulum in this specimen, the uncinate process is short and placed low down, while the remnant bulia hangs from the med. turbinate.

(D) This sinus is unusually high up on the maxilla. The first puncture into the inf. meatus went into the soft tissues of the cheek. Schaeffer has demonstrated this point well and has seen a specimen.

(E) These two specimens have a band for the uncinate process; the infundibulum of which forms the inferior margin of the hiatus, while below it is another opening leading into the infundibulum and the antrum.

(F) The accessory opening was high up and classed as an adjoining ostium; in 9L. it was low down, larger in size and was called an accessory ostium. Some might call both of these accessory ostia; 9L. had in addition a distinct accessory ostium.

roof was 13 mm.; to this must be added the length of the housing of the electric lamp, distal to the objective lens, a distance of 12 mm., which makes the total 25 mm. With this total must be figured (a) the number of millimeters that the canine fossa puncture lay above the floor of the sinus, plus (b) the possibility that the abutment of the tip of the antroscope against the superior wall or roof of the sinus may not be exactly the highest point in the sinus. Roughly, it seems rational that the difference of 25 and 33 or 8 millimeters would be found in these two small factors—i. e., these measurements for the inferosuperior dimension of these specimens are about average size. Also, the mediolateral dimension of the antra in Schaeffer's 150 specimens measured 23 mm.; the average for these thirty-four were 4.56 mm., plus 12 mm. used up by the antroscope bulb, making roughly an average of 16.5. The difference of 6.5 mm. is explainable by the fact that the puncture unfortunately does not pass through the widest dimension of this direction—but rather posterior to it and also at a slight angle to it. In short, I feel that the antroscopic measurements check well with normals, and the specimens were average, even if this can only be estimated and crudely shown.

There is a small distinct difference between the right side and left side in the measurements of all these antra, as can be seen in Table II, the right side being the larger. On the other hand, the number of specimens is small and I think that one must disregard this conclusion for that reason.

The question as to whether one will see much of the antral mucosa is not entirely dependent upon the length that the objective lens gets into the antrum, but rather, to a great extent, upon the conformation and shape of the antrum. For instance, specimen 13R. permitted the objective lens to enter 8.5 mm. through the meatal puncture and the ostium was not even in the field of vision. Whereas, specimen 15R., with only 2 mm. of lens in the antrum, had a fair view of the antrum and permitted one to see two ostia. Similarly, antrum 2L. allowed one to see the ostium when the lens was only 2.5 mm. in the antrum. A more evident case is specimen 3R., in which the lens passed through the canine fossa for 24 mm. and could not view the ostium because of a small

infra-orbital recess. There are several more such comparisons, as can be seen on studying Table II.

The above paragraph indicates that if we are to make antroscopy practical and more general we will need an objective lens that can see into recesses and one that can be directed from an exceedingly small range and limitation of movement to view all areas. In short, we need an instrument that will have the lens at the tip or point of the nasopharyngoscope. As mentioned, the nasopharyngoscope or antroscope used in these examinations has a housing for an electric bulb which measures 12 mm. from its distal tip to the objective lens or "eyesight" of the instrument. To reiterate, before one can begin to view the inside of the antrum there must be at least a dimension of 12 mm. or more from the point of puncture into the antrum to the opposite wall or diagonal, since this wall stops the further entrance of the antroscope and its lens. There are on the market at present nasopharyngoscopes that measure less than 12 mm. from the distal tip of the electric bulb or housing to the objective lens. In fact, the best known to me measures 10 mm., which means a bigger view and more certainty of seeing into the antrum, not of merely seeing 2 mm. more of the antral wall. However, this particular last mentioned instrument has other objections.

It should be brought out that for every initial millimeter more that the objective lens can enter into the antrum or a recess, it multiplies several times the amount of lining one can see. This last is dependent upon how far the walls are from the objective lens and the angle of vision which the lens is permitted. The greater the distance that the wall is from the lens, the greater the amount of surface that can be seen. Physically, the amount to be seen is the result of the angle permitted the objective lens to view times the square of the distance of the viewed wall from the objective lens. To quote from the previous article on this subject:⁸

"The space within the antrum is small and limited, while the distance from the distal tip of the instrument to the observing lens is more than 1 cm. This extra length prevents the antroscope from penetrating deeper into the sinus; that is, the observing lens, which is the "eye" of the instrument, may be just within the an-

TABLE II.

Specimen Number	RIGHT SIDED ANTRIA				LEFT SIDED ANTRIA			
	Length of Total Entry	Was Ostium Seen	Length of Canine Entry	Was Ostium Seen	Length of Medial Entry	Was Ostium Seen	Length of Canine Entry	Was Ostium Seen
1.	8. mm.	no	11.5 mm.	yes	4. mm.	no	9.5 mm.	yes
2.	2. mm.	no	14. mm.	yes	2.5 mm.	yes	13.5 mm.	yes
3.	7.5 mm.	yes	24. mm.	no	4. mm.	no	17. mm.	no
4.	6.5 mm.	no	13. mm.	yes	9. mm.	yes	19. mm.	yes
5.	3.5 mm.	yes	5. mm.	yes	5. mm.	yes	15. mm.	no
6.	3.5 mm.	yes	13. mm.	yes	2. mm.	no	14. mm.	no
7.	1. mm.	no	11. mm.	no	3. mm.	no	5.5 mm.	no
8.	11. mm.	yes	29. mm.	yes	4. mm.	no	19. mm.	yes
9.	3. mm.	yes	19. mm.	yes	6. mm.	yes	12.5 mm.	yes
10.	3. mm.	yes	11.5 mm.	no	2.5 mm.	no	12.5 mm.	yes
11.	6. mm.	no	8. mm.	no	2.5 mm.	no	5.5 mm.	yes
12.	0. mm.	no	10. mm.	yes
13.	8.5 mm.	yes	18. mm.	yes	1.5 mm.	no	11. mm.	yes
14.	4.5 mm.	yes	15.5 mm.	yes	9. mm.	yes	11. mm.	no
15.	7. mm.	yes	7.5 mm.	yes	5. mm.	no	12.5 mm.	yes
16.	3. mm.	no	17. mm.	yes
17.	1. mm.	no	9. mm.	yes	4.5 mm.	yes	17. mm.	yes
18.	no	yes	9. mm.	yes	15. mm.	yes
19.	8.5 mm.	no	19. mm.	no	7 no	4 no
20.	9 no	3	7 yes =	10 yes =
Total	82.5 mm.	46.6 % positive results	228. mm.	82.3 % positive results	72.5 mm.	50 % positive results	212. mm.	71.4 % positive results
Average length of entry	4.85 mm.		13.41 mm.		4.26 mm.		12.46 mm.	

trum or may not be within the antrum at all. The distance from the distal tip to the observing lens or prism should be lessened; this is a simple matter for an instrument maker. With the length cut down, a greater portion of the antrum will be seen."

Unfortunately, cutting down the distance from the distal tip to the viewing lens evidently is not a simple matter for the instrumentmaker. It is possible to have made an instrument which will cut this down to 9 mm. in addition to some minor improvements on the present antroscope, but I feel certain that this not what is needed. I have designed an instrument which I think will serve—i. e., one with an objective lens at the tip of the "scope," but it requires experimentation in its making, and the instrumentmakers are avoiding that in these times.

To compare how much of the antrum is seen through the inferior meatal route as against that of puncture through the canine fossa is a somewhat difficult question to settle. The anatomical configuration, the possibility of recesses and ridges, and the chance of a fragment of bone fractured off by the puncture obstructing the view are material determinants as to the extent of the antrum to be seen. Finally the distance that the objective lens may enter into the antrum is one of the foremost factors as to how much of the mucosa will be seen. Considering all these factors, I think one may form some gross general conclusions on comparing the observations in Table I.

In a rough way one has a chance to probably see more of the mucous lining in going through the canine fossa than through the meatal route. However, one can glance through Table I and find several exceptions—i. e., in which the meatal puncture permits more of the lining membrane to be seen. In examining Table I one can see that our present equipment does not give consistently satisfactory results in either passage. It is not a question of the most desirable passage but definitely one of requiring improved equipment. It is soon apparent on comparing several specimens that even though the canine fossa puncture permitted the lens to enter an average of 8.44 mm. more than the meatal route as figured from Table II; yet one may see more with a less covered distance through the meatal puncture. This is sharply shown in case 3R., which permitted the lens of the scope to be passed

through the canine fossa for 24 mm., and it was impossible to see the ostium, while in case 15R. it passed only 2 mm. through the meatal puncture and the ostium was seen. The same point is true between 15L. and 2L., and in several other cases to a less distinct degree. In other words, the meatal puncture is in a better position to determine the pathology of at least the ostium, floor and probably the whole sinus. However, this route permits less side motion and forward extension of the nasopharyngoscope. With the present instrument one will see more, usually, by puncturing through the canine fossa, though exceptions to this rule are certainly not rare.

Fragments of bone obstructing vision, particularly of seeing the ostium, occurred only in puncture through the inferior meatus; it never happened in going through the canine fossa. From a theoretical attitude one may explain this on (a) the angle of the trocar against the plane to be punctured is more oblique in the inferior meatal course; (b) the bone is usually thinner in the meatal passage; (c) that fragments occur just as frequently in the canine fossa but not being sought for, since they cover no important structure, they are not seen; (d) finally, the periosteum and soft tissues around the canine fossa serve to hold or prevent fragments of bone from breaking off and going into the antrum. Whichever of these reasons are accountable matters little. The fact seems that one does not have fragments of bone obstructing the view in the antrum on puncturing through the canine fossa, while this does happen in the passage through the inferior meatus, as in 11L., 16R. and 20R.

In contrast to these advantages for puncturing through the canine route it should be pointed out that puncturing through the canine fossa is usually through thicker bone and commonly requires a mallet. In addition, there is the necessity of a more certain anesthesia and injection of some local anesthetic into the tissues for puncture through the canine fossa. In puncturing through the meatal passage one rarely requires a mallet, and anesthesia is readily obtained by topical application of cocain.

The clinical value of being able to see the mucous lining of the antrum is quite apparent. Foremost, it gives the otolaryngologist almost immediately the extent of the disease or pathology. Our

present conceptions of pathology and diagnosis of the sinuses are somewhat limited because we do not see the fresh living membrane. Practically the only time when this opportunity is possible is when one does an external or radical operation on the sinus, and this is assumed to occur only in a thoroughly diseased lining. If we want to be more sure of the most efficacious treatment, accurate and more certain diagnosis will be the means. This is possible by seeing and if possible palpating the lining membrane of the sinus rather than our indirect methods of roentgenology, washings, transilluminations, etc. This point cannot be too well emphasized, for we have been fixed to these established methods as the only form of diagnosis. Furthermore, before us is the possibility of treating maxillary sinusitis intra-antrally by means of a puncture only.

Some men have tried to go further with the use of the nasopharyngoscope as an aid in treatment. Morse⁷ has used the nasopharyngoscope to implant radium seeds into polypoid tissue in the ethmoid region. More recently, Trotter⁸ has successfully treated ten cases of chronic maxillary sinus with diathermy and the antroscope. In these cases only a puncture was made into the canine wall, for the antroscope to view the coagulation, and a window from the nasal fossa into the antrum, for the coagulating electrode. This certainly would appear to be a highly desirable method of treatment as compared to our present method for removal of polyps, etc., by an external maxillary sinus operation. But we are far from our goal of treating disease by means of or with the aid of the nasopharyngoscope. I feel that most of our delayed progress is due to the type of instrument we use, and not to any lack of desire to go forward on the part of the otolaryngologist.

PROBLEM NO. II.

The second problem of which route permitted the ostium to be seen more frequently is dependent to some extent on the above first question of how much of the antral lining can be seen. Of the seventeen right antra punctured we see from Table II that the ostium was in the field of vision on puncturing through the inferior meatus in eight cases and that it could not be brought

into the field in nine cases, whereas, the puncture through the canine fossa brought the ostium in view in fourteen cases and was impossible in three cases. In percentages it means the ostium could be seen in 46.6 per cent of cases through the meatal passage and 82.3 per cent through the canine fossa. Of the left antra these figures were: Through the meatal passage seven could be seen out of fourteen, or 50 per cent, and the means of the right and left groups of antra is through this passage 48.3 per cent. Similarly, the left canine fossa permitted ten out of fourteen cases, or only 71.4 per cent. The means of the percentage for canine fossa is 76.6 per cent, as against 48.3 per cent of the meatal passage, or, if considered as one group of thirty-one antra, the percentages are 77.4 to 48.3 per cent.

There are two factors that should be mentioned here in relation to Table II. One that there was a slight adjustment made in Table II, to point: In the meatal puncture through specimen 2R. the ostium was seen only on marked shifting. This was possible because no nasal septum was present. In the meatal puncture through specimen 14R. the ostium was seen in its lower half only. These two observations were considered as one positive result, namely, 2R., while 14R. is marked negative. The second factor of significance is that many of the specimens were crania cut in the sagittal section and had no nasal septum, and this permitted a more perpendicular puncture into the inferior meatus than would be possible with a septum to limit the angle of puncture in the inferior meatus. Also, the traction of the cheek in puncturing the canine fossa was slightly more than would be usually done in a patient. In other words, the percentages obtained in these punctures are high, and better than would be gotten in a similar number of like patients.

Maxillary ostia are defined as either normal or accessory ostia, depending upon whether these ostia are located in the infundibulum or outside of this gutter. These last are classed as accessory ostia and are usually found inferior to the hiatus semilunaris, while the former are the normal ostia. Schaeffer and Davis describe another group known as "duplicated" or duplicate ostia, which are defined as an additional or duplicate ostium in the floor of the infundibulum. Schaeffer writes: "Dupli-

cation of the ostium maxillare is a condition in which the sinus maxillaris has two ostia, both of which communicate with the infundibulum ethmoidale." If one will study Table I carefully he will find that there is at least another group not defined by any of these conditions and that the type classed as duplicate ostia is of two varieties.

Duplicate ostia may be found to be as two distinct openings in the floor of the infundibulum with each having its own ostium or passageway into the antrum. This is one of the two types of duplicate ostia, or perhaps better called, normal duplicate ostia, and is seen in specimens 7L. and 9L. The second type is to have two infundibular openings joining to form a single common opening into the antrum as in case 14L., or reversely, and what I think is more rare one infundibular opening developing to two openings in the antrum, as in case 11R. These might be called adjoining ostia, or more exactly, adjoining normal ostia. It is these adjoining types of ostia that make one visualize and appreciate that our so-called ostia of the maxillary sinus are really canals, not "windows."

The next type which seems to be unnamed or not classed is to have both a normal and an accessory ostium join to form a single common opening into the antrum. This type may be called normal accessory adjoining ostia. It was a surprise to find that in this small group of thirty-four specimens there were two of this type, namely, 9R. and 12R.

One may continue this basis of classification, calling two distinct and separate accessory ostia as accessory duplicate ostia (this type is not rare), and finally with two accessory ostia having a common opening into the antrum as accessory adjoining ostia, this is probably a very rare specimen. To conclude, one may classify maxillary ostia as follows: (1) Normal ostium; (2) normal duplicate ostium, as 7L. and 9L.; (3) normal adjoining ostia, as 11R. and 14R.; (4) normal and accessory adjoining ostia, as in 9R. and 12R.; (5) accessory ostium, as 5L.; and also (6) accessory duplicate, and (7) accessory adjoining ostia, of which there are none in this group.

The clinical value of being able to see the ostium in cases of questionable or definite sinus disease would seem to be worth

while. It would immediately inform us as to whether it is the slit-like type, which is probably obstructed or occluded easily by inflammation and reaction, or thickened, discharged or foreign body; or whether there is a large patent orifice sufficient for drainage, although it might be in a poor situation for dependent drainage. This point will be brought up again later on. It should be mentioned here that accessory ostia were not usually seen by antral puncture. However, they are readily seen if in the typical position of posterior or inferior to the hiatus semilunaris by ordinary nasopharyngoscopic examination of the nasal fossa. When present they should certainly be the route for washing or flushing when this is the procedure elected, as has been mentioned before by Schaeffer⁹ and many others.

Following the antroscopic observation of these antra, they were examined directly—that is, if either the anterior or posterior wall of the sinus was not already open, it was then removed so as to give a complete inspection of the antrum in order to check over the results of the antroscopic notation and to permit carrying out of the final problem. This readily disclosed errors in the antroscopic observation. These are, for the most part, of the following type:

Not recognizing an ostium when it is in the field of vision, as in cases 3RM., 8RM. and 8RC., 12RC., 15RC.; 15LM. (M. = meatus; C. = canine). This is due to a slit-like type of ostium, being quite narrow and at times difficult to discern, especially if the membrane is irregular and in folds. It is this type of opening that probably closes easily and has a tendency to be seen with diseased membrane, because of limited aeration and drainage. Here perhaps should be mentioned missing of an adjoining or accessory ostium—having seen one ostium, the tendency is to be satisfied and not look for the second, which may be actually in view. I thoughtlessly made this error several times. One must continually be on guard against it.

Missing definite pathology, such as polyps, as in cases 2R. and 2L., in these cases were due to my not being prepared to see them. However, there is also the possibility of not seeing polyps, etc., because of the limited view permitted by the present instrument.

Mistaking a ridge in the antrum as the opening into a canal,

which happened to me in 5RC., 14RC. and 18RC.: This is due to the inability to see above the ridge—i. e., explore the antrum above this shelf. It is unusually common to have a fold of mucosal lining, at times including a bony ridge, project out from the margin of the ostium, as will be mentioned later. It is this appearance which deceives one into thinking of an ostium. However, if one could explore or see above the ridge he would rarely make this mistake.

PROBLEM NO. III.

We call the opening of the maxillary sinus into the infundibulum the ostium maxillare. Dorland's Medical Dictionary defines an ostium as: "A mouth or orifice. Ostium abdominale tubal uterinæ (B. N. A.), the fimbriated end of an oviduct," etc. Stedman's Medical Dictionary defines ostium: "(L. Dim. of os, mouth.) A small opening, especially one of entrance into a hollow organ or canal," etc.

It was my expectation and conception that in antroscopic examination I should be able to get a full view of the maxillary ostium. Since there was no canal, I should be able to see out to the hiatus semilunaris and in some few cases into the mid. meatus. However, on examining several antra this way, I realized that I would rarely be able to see through this so-called maxillary ostium: for it was really not an ostium but a passageway—i. e., a canal or duct, in most cases. In Gray's Anatomy,¹⁰ the editor does not use the term ostium but calls it an opening. However, in illustrations 880 and 881 he labels this opening an ostium, and both of them appear to converge as if into a short canal. All he says concerning the ostium is: "In the anterosuperior part of its base (i. e., medial wall) is an opening through which it (the antrum) communicates with the lower part of the hiatus semilunaris." The basis for most early anatomic descriptions of the accessory sinuses is from Zuckerkandl.¹¹ The following is a translation of his description of the maxillary ostium: "Its form in every position is not established, since the opening out from the infundibulum is not clearly presented. To survey the whole circumference around the opening it is advisable to observe it from the sinus maxillaris to here. By such a procedure it is shown that the

ostium maxillaris rests just under the orbital floor, lying before the prominentia lacrimalis, and the relationship between the size and form varying to some degree. The foremost shape is an elliptical slit, with its longer axis directed sagittally; in many cases, however, it is almost circular or through pressure the border is almost kidney shaped.

"The smallest openings which I met in the greatest proportion were roundly formed and had a diameter of 3 mm.; the largest covered a length of 19 mm. and a width of 5 mm. Between these two extremes are to be found cases varying between the length of 7 mm. and 11 mm., the width between 2 mm. and 6 mm. At times it is divided by a band of mucous membrane in two parts."

Most writers assume that there is no length—i. e., mediolateral—to this so-called ostium maxillare, and only describe one or two dimensions of the opening in its sagittal plane. For instance, A. Logan Turner¹² writes: "The size of the opening varies. It may measure only 2 by 3 mm., or it may have an anteroposterior dimension of 18 mm. When it reaches a considerable size it may almost entirely replace the outer wall of the infundibulum and form a long slit-like communication between the antrum and that channel."

Parsons Schaeffer⁹ gives a thorough, detailed description of the connection between the ostium of the maxillary sinus and the nasal fossa:

"The maxillary sinus communicates indirectly with the meatus nasi medius by means of a series of openings: (1) The maxillary ostium, which pierces the superior and ventral part of the base of the sinus to open into (2) the infundibulum ethmoidale, thence via (3) the hiatus semilunaris into the meatus nasi medius. It must be kept in mind that the ostium is located in the superior part of the sinus and that it opens into the infundibulum ethmoidale and not into the hiatus semilunaris, as many writers say. The maxillary ostium is located either in the most dependent part of the infundibulum or in the lateral wall of this channel, and varies from 1 to 12 mm. in distance from the hiatus semilunaris. The distance is dependent upon the width of the processus uncinatus and the resultant depth of the infundibulum ethmoidale at this point."

Schaeffer states that the distance from the margin of the hiatus semilunaris to the floor of the infundibulum or the depth of the infundibulum may be 1 to 12 mm. In the table above it is rarely 1 mm.—it usually averages 6 to 8 mm., which is about what Davis finds them to be.

Schaeffer continues: "The maxillary ostium may be round, but as a rule is either oval or elliptical. In a series of 100 cases examined by the writer it had a great range of dimensions, varying from 1 to 22 mm. in length and from 1 to 6 mm. in width. In cases where the ostium reaches a considerable size it may entirely replace the lateral wall or floor of the infundibulum ethmoidale, thus forming a long, slit-like communication between the maxillary sinus and the infundibulum ethmoidale."

Warren B. Davis¹³ writes: "Communication between the sinus maxillaris and the meatus medius is via the ostium maxillare, the infundibulum ethmoidale and the hiatus semilunaris. The distance between the ostium maxillare and the hiatus semilunaris varies in adult cases from 4 to 12 mm., according to the width of the processus uncinatus and also to the degree of the medial inclination of its free or posterosuperior portion."

Hajek¹⁴ describes the opening as follows: "The form and size of the ostium maxillare vary. It usually forms an elliptical cleft with its long axis directed sagittally. It is rarely circular or kidney shaped. The size varies from an opening 3 mm. in diameter to an opening 19 mm. in length to 6 mm. in width. When the opening is greatly elongated the whole hiatus semilunaris may open into the antrum."

All these authors have called and give dimensions for the opening of the maxillary antrum into the infundibulum as a simple ostium or window, having dimensions only in the sagittal plane—i. e., anteroposterior and inferosuperior direction.

However, Skillern¹⁵ distinctly and definitely described a canal for this maxillary opening into the infundibulum, even though he calls it an ostium.

"It may assume one of several forms: Round, oval, kidney-shaped, or in the nature of a long slit. The oval form, however, predominates. The size varies from 2 mm. to 1.7 cm. by 1.1 cm., the average ranging from a buckshot to that of a pea. This open-

ing (ostium maxillare) is not situated as a window in a wall communicating directly from within outward, but takes a distinct direction upward, backward and inward. From the nasal aspect the direction is naturally downward, forward and outward."

The direction that Skillern gives for this opening, or better, for the canal is true in the majority of the cases but not for all of them. It should be mentioned here that the direction of the ostia or canals given in Tables I and II is always considered from the nasal fossa into the antrum.

The distance between the ostium maxillare and the hiatus semilunaris margin, Davis said, "varies in adults from 4 to 12 mm." The extremes in these thirty-two antra, as shown in Table III, were 4 mm. in specimen 7L., or perhaps zero in 3L., and 13 mm. in 1L. and 7L. The average for the twenty right ostia was 8.33 mm. for this same distance, and for fourteen left specimens it was 8.64 mm., or for the total thirty-four specimens as one group the average distance from the border of the hiatus semilunaris to the internal margin—i. e., the antral margin of the ostium maxillare—is 8.45 mm.

The distance from the floor or lateral wall of the infundibulum to the antral margin of the so-called ostium maxillare (mediolateral dimension of ostial opening) varies from 1 mm., as in cases 8L., 15R. and 20R., etc., to 9 mm. in 7L. The average for twenty right ostia for this same distance was 4.53 mm. and for sixteen left ostia 4.34 mm., which are fairly close, particularly for such a small number in each group. The average distance for the length of this canal or duct, or as it is commonly called, ostium, for thirty-six normal ostia is 4.44 mm. In those cases which had one wall of the canal longer than the other the length of these walls were averaged and the means taken as the length of the passageway.

In this group of cases only normal ostia were included. This, of course, includes what are called duplicate ostium. The structure of these duplicated ostia vary somewhat. However, these specimens usually have a single opening into the infundibulum with two passageways into the antrum—i. e., the adjoining ostia mentioned above. These may have only a band of mucous membrane separating them or also include a ridge of bone between

them, and at times there may be quite a distance between them—i. e., another fairly common type was to have two distinct and complete passageways from the antrum into the infundibulum. The uncommon type of adjoining ostia is to have a double opening into the antrum as occurred in 11R. These duplicate or adjoining ostia should help one to visualize and readily prove that these so-called ostia are not "windows" but canals, since there must be formed some sort of wall in order to produce two openings into one—i. e., there must be length to these passageways.

It is not uncommon to find an accessory ostium having a passageway—i. e., really a canal. This is shown in the accessory passageway in the above mentioned normal accessory adjoining ostia, as in case 12R., where a valvelike flap existed and made a canal of 10.5 mm. length, and of 9L., which had a canal of 7 mm.

Embryologically the frontal and maxillary sinus arise from ethmoid cells. The appearance of a nasofrontal duct depends, as Schaeffer shows, on the development of the frontal recess and which of these cells finally form the adult frontal sinus. In clinical language, a nasofrontal duct is said to be present when there is present an anterior overhang to the middle turbinate which completes the rather indefinite passageway from the frontal sinus into the middle meatus. In the case of the maxillary sinus there is really always a duct or canal connecting the maxillary sinus with the infundibulum, as will be understood from Schaeffer's description of the origin of the maxillary sinus and its opening.

"The location of the maxillary ostium of the adult corresponds to the place of the primitive maxillary pouch. The pouch gradually develops into the pyramid-shaped cavity of the adult, leaving the place of communication with the infundibulum ethmoidale at the primary evagination."

However, because of the proximity of this maxillary sinus to the infundibulum this canal or duct may be so decreased as to measure as little as 1 mm. In these cases the canal has been "squeezed" so short as to measure 1 mm.—i. e., the sinus is enlarged so that the opening which originally and usually is a passageway having some mediolateral dimension, has been greatly decreased to a "window." The criterion as to whether this open-

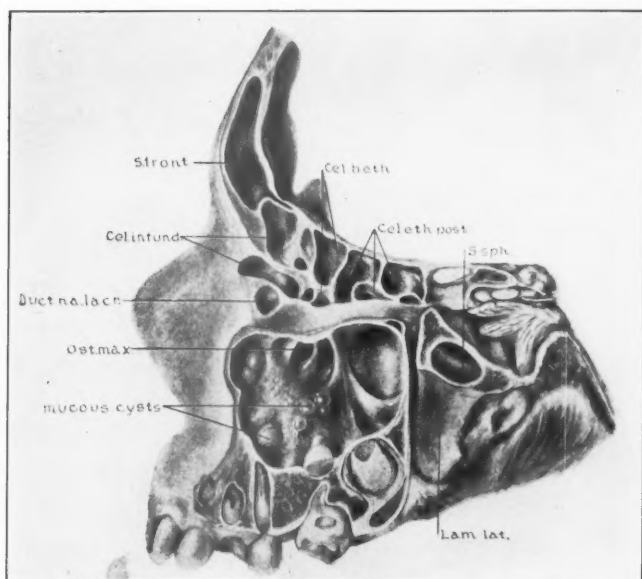


Fig. 2. This illustration from Davis' "The Development and Anatomy of the Nasal Accessory Sinuses in Man."

ing is to be called a canal or a window is really the length of the mediolateral dimension of this passageway.

In reading the various texts mentioned in the bibliography one can see illustrations of the antrum in which the artists have actually drawn the maxillary ostium as a lumen leading into a canal, and in some few cases one may see a frontal or coronal section showing the whole length of this canal rather than a thin window, as is shown in Figs. 3 and 4.

Dr. Warren B. Davis has been so very kind as to loan me from his book, "The Development and the Anatomy of the Nasal Accessory Sinuses in Man," one of his illustrations, Fig. 2 (labeled Fig. 47 in his book). In it the artist has plainly drawn, for the maxillary ostium, what appears to be a lumen leading into a canal. In addition, this illustration seems to show another opening posterior to the first, which demonstrates a duplicate, or

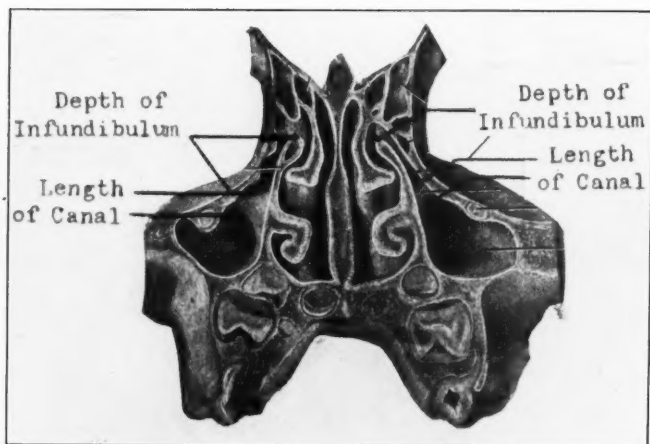


Fig. 3. This illustration taken from "Anatomy of the Nasal Accessory Sinuses in Infancy and Childhood," by Warren B. Davis.

better, an adjoining ostium, the latter one also appearing to be a canal. Another illustration in this same text showing a canal is Fig. 37, which is a coronal section passing through the infundibulum, and the so-called ostium maxillare. Here one sees a canal leading into the infundibulum which in itself simulates a canal. However, one readily recognizes that the passageway from the antrum to the infundibulum is a canal of some length and not a mere window.

Schaeffer also has several specimens in his book in which the artist has drawn a canal for the ostium, namely, Fig. 83 and Fig. 89, on the right side. Skillern in his book has an illustration, Fig. 48, showing duplicate ostia, both of which look like canals, while Fig. 50 shows the normal ostium with a distinct lumen indicating a canal.

The criterion as to what constitutes an ostium or a canal is naturally the length of the opening. I think a passageway that is longer than 2 or 3 mm. should be recognized as a canal.

The direction of these canals is not always definite, since the width and height of the canal may be large—i. e., the walls may

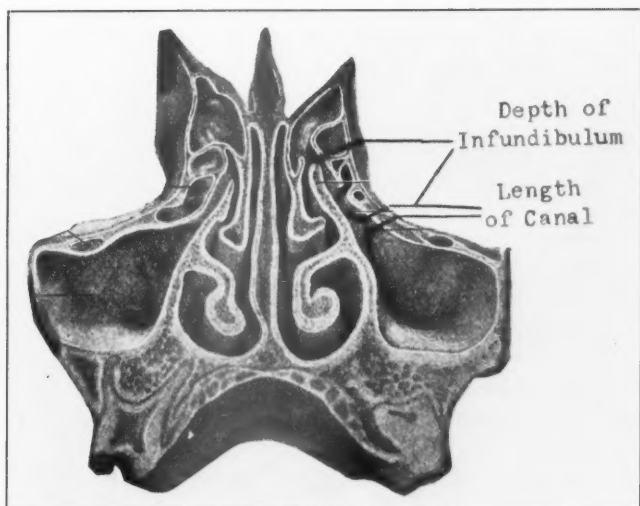


Fig. 4. This illustration taken from the "Anatomy of the Nasal Accessory Sinuses in Infancy and Childhood," by Warren B. Davis.

be wide apart and exert no pressure for direction on the course of the probe. But usually the canal does direct the probe in a certain course, though in some instances there may be some latitude in the direction. Another factor concerned is the surrounding structures—i. e., bulla and uncinate process—which in some cases tend to affect to some degree the relative direction of this canal. Recognizing these facts, I thought that the direction a bougie would take in probing this passageway would best be classed in only two main groups. The first would include all specimens whose canals sloped in any one or combination of the following directions, anteriorly, laterally or inferiorly; while the second class would consist of those canals which were directed posteriorly or in a combination of this direction with that of an inferior or lateral course. With this basis of classification of the twenty right side ostia, twelve, or 60 per cent, were of the first class and 8, or 40 per cent, were of the second type. Again, of sixteen light ostia there were eleven, or 68.75 per cent, of the first group, and five, or 32.25 per cent, of the second variety.

Finally, taking all the specimens as one group of thirty-six ostia, there were 64 per cent of the first class and 36 per cent directed as in the second class of ostia.

The clinical value of knowing whether there is a canal or a window to drain the maxillary antrum is but limited. When one realizes that the passageway for emptying the antrum is a canal the possibility for its closure is more evident. Zuckerkandl¹¹ writes:

"Die anatomische Desposition für einen leichteren oder schwereren Verschluss spielt aber in erster Reihe keine Rolle, denn durch krankhafte Prozesse der Highmorshöhle wird die Öffnung nur selten verschlossen; Ich habe dies bisher nur einmal beobachtet."

This would seem to me an exceedingly low occurrence for closure of this passageway. In my limited experience I have had a patient both of whose maxillary antra were always difficult to force normal saline through when the sinus was punctured through the inferior meatus and in whom it was frequently impossible with fairly strong pressure to force any fluid. (The punctures were always made in the inferior meatus.) There was little doubt as to the closure of both of the maxillary sinus natural openings occurring on and off over a period of months. In specimen No. 7R. I was unable to find an ostium on the first examination, even after the anterior and posterior walls were removed. On going over this specimen again, I was able to detect what looked like an opening, and with a most narrow, soft probe I was able to put it through this passageway. Surrounding this opening were two small sessile polypi. I feel that this passageway closed many times during life, and that aeration and drainage were always limited with a passageway of this description. It is readily understood that the length of this passageway is not the only factor that accounts for its closure. The height and width of this canal is equally as important in cases of closure as the length. In short, all three dimensions are factors determining whether the opening will close or not. However, my experience is quite limited and I would like to hear from older and more experienced men as to the frequency of closure of the so-called maxillary ostium.

A second clinical factor concerned in the presence or absence of the canal is in the possibility of probing it—i. e., catheterizing of the so-called natural ostium. Many well recognized men have felt that this is an unusually difficult task—in fact, in some cases impossible. A. Logan Turner⁷ writes: "The concealed position of the ostium behind the uncinate process, the prominence of the bulla and the small diameter of the hiatus semilunaris in many cases are factors which make successful catheterization of the ostium a difficult and not infrequently impossible procedure."

Hajek¹⁴ writes, more mildly: "In a majority of cases the difficulty of sounding the ostium maxillare, regardless of its size and variations, lies not so much in its narrowness as in the hidden location in the depth of the infundibulum and in its anatomic relations in the middle meatus."

Schaeffer⁹ writes: "An examination of a large series of specimens leads the author to believe that it is impossible clinically, in the vast majority of cases, to sound the maxillary sinus through its normal ostium," etc.

From a consideration of Table III, it would seem that catheterizing the opening into the maxillary sinus would be difficult in most cases and impossible in some. In addition to the several factors brought out above, there are the following: (1) The so-called maxillary ostium is usually a canal, not a mere window—i. e., this opening must be blindly followed for an average of 4 or 5 mm. in order to be in the sinus, to say nothing of the difficulty of finding the floor of the infundibulum and then being able to palpate the infundibular opening into the maxillary sinus. This last seems extraordinarily difficult because of the overhanging bulla and pressure of the lateral wall of the middle turbinate. Again, as shown in Table III, the direction of these canals varies. In a rough way, 64 per cent follow roughly the direction of Skillern, anteriorly, inferiorly and laterally, while 36 per cent, roughly, go posteriorly or posteroinferiorly and laterally, or in a combination of these. At times it was quite difficult to probe some of these canals, even after the middle turbinate was removed, and I saw and entered the opening in the floor of the infundibulum leading into the sinus. Simply because of a wrongly directed bougie, pressure being exerted against the wall of the canals in-

TABLE III.

Specimen Number	RIGHT SIDED ANTRA			LEFT SIDED ANTRA		
	Undulate Border to Ostial Margin	Length of Canal	Direction of Canal	Undulate Border to Ostial Margin	Length of Canal	Direction of Canal
1.	10. mm.	6. mm.	Ant., inf.	13. mm.	6. mm.	Inf., lat.
2.	8.5 mm.	6.5 mm.	Inf., lat.	6. mm.	2. mm.	Inf., lat.
3.	10.5 mm.	7. mm.	Ant., inf.	4. mm.	Ant., inf.
4.	6. mm.	2. mm.	Laterally	7. mm.	3. mm.	Anteriorly
5.	10. mm.	6. mm.	Inferiorly	11. mm.	8. mm.	Post., inf.
6.
7.	11.5 mm.	8. mm.	Inf., post.	13. mm.	9. mm.	Inf., lat.
8.	6. mm.	2. mm.	Inf., post.	6. mm.	1. mm.	Ant., inf.
9.	7.5 mm.	2.5 mm.	Post., inf.	3. mm.	3. mm.	Inf., post.
10.	9. mm.	3.5 mm.	Post., inf.	1.5 mm.	Laterally
11.	10. mm.	7. mm.	Post., inf.	10. mm.	4. mm.	Inf., lat.
12.	8. mm.	4. mm.	Inf., post.
13.	10. mm.	3. mm.	Posteriorly
14.	7. mm.	3. mm.	Laterally	8. mm.	6. mm.	Lat., ant.
15.	9. mm.	3. mm.	Lat., inf.	8. mm.	3. mm.	Posteriorly
16.	6. mm.	4. mm.	Ant., inf.	10. mm.	6. mm.	Laterally
17.	Inf., post.
18.	9. mm.	4. mm.	Post., inf.	7. mm.	2. mm.	Inf., post.
19.	5. mm.	1. mm.	Inf., post.	9. mm.	5. mm.	Lat., post.
20.
Total	166.5 mm.	90.5 mm.	121. mm.	69.5 mm.
Average Length	8.325 mm.	4.525 mm.	8.643 mm.	4.343 mm.

stead of following the sides, at times it required several shiftings of this direction to finally negotiate the passage.

Naturally, I feel that in a patient and the presence of a middle turbinate, the task is much more difficult and at times impossible. I think that when the catheter enters the sinus it often pierces the membranous portion of the sinus wall, at times fracturing the uncinate process. On the other hand, the normal opening may be also catheterized. The immediate damage to the surrounding tissues when the membranous portion is punctured is probably less than that caused by entering through a puncture in the inferior meatus. I feel that this is so except for one point. Proetz has frequently and well brought out the great significance of ciliary function in draining the sinus. I wonder if not more harm and delay to normal drainage is produced by injuring ciliary function in and about the area of the draining passageway or so-called ostium, as compared with traumatizing and destruction of some localized limited area of mucosa lower down on the medial wall of the sinus.

Certainly there is nothing new in this viewpoint of feeling that catheterization of the so-called natural ostium or orifice, because of location of the infundibular opening and surrounding anatomy, is in the majority of cases not easy. It is new, however, or makes us admit that the procedure is still more difficult if one recognizes that the natural opening to the maxillary sinus is not an ostium or window but rather (1) a passageway or canal, and still more so when one realizes that the (2) direction of this canal is not set but takes a variable direction or course, which is unknown beforehand to the operator; and (3) that the catheterizing instrument is a fine metal tube and has its own direction or course, which is the only one that it can navigate or catheterize.

Just recently Myerson¹⁰ wrote the following in an article advocating catheterization of the natural orifice of the antrum:

"Recently Dr. Marvin Jones called my attention to the fact that he had noticed a nasomaxillary passageway in some of his films made following the injection of iodized poppy seed oil, 40 per cent. With this in mind, I reviewed the collection of Dr. Herman Rubin and found several such passageways, which are outlined in the accompanying illustration."

This certainly is more proof of the presence of an infundibulo-maxillary canal. I wish to say that this is my first knowledge of this statement. The above paragraph was given in a paper delivered at the 1931 A. M. A. meeting, which I happened to attend. I unfortunately missed this paper and neither read nor heard the view of the above paragraph.

Personally if anyone wants to check the presence of a canal from the antrum, I would suggest doing it directly on the cadaver with a graduated probe—not by opaque oils and roentgenographs, which are an indirect method. Further, it introduces a possible factor of error, in that the oil may drop to the inferior hollow of the infundibulum, making it appear as a part of the infundibulo-maxillary passageway.

CONCLUSION.

Three problems were undertaken and the results of these are:

1. In puncturing the maxillary sinus with a nasopharyngoscope or antroscope through the canine fossa (i. e., facial wall) or the inferior meatus, one has in general a better chance of seeing more through the first or canine route. However, the question of seeing the important structures varies much and quite frequently one will see more by using the inferior meatal route. The main point, I feel, is that the type of instrument used at present is at fault, it mattering little which route is used, and that a different principled instrument is needed.

2. The ostium maxillare will be seen more frequently by puncture through the facial wall of the sinus, as against puncture through the inferior meatal route; in a group of thirty-one specimens this proportion was 77.4 per cent as against 48.3 per cent positive results.

3. The mediolateral dimension of the so-called ostium maxillare, as averaged for thirty-six normal ostia, was 4.44 mm.; that this passageway for the average case was a small canal and should more exactly be called in such cases the infundibulo-maxillary canal rather than the maxillary ostium.

This canal has a variable direction and in a crude way flows from the infundibulum into the antrum in one or a combination of three courses of anterior, inferior or lateral in 64 per cent of the

cases and of posterior, inferior or lateral in 36 per cent of the cases of thirty-six ostia. The types of this infundibulomaxillary canal are of greater variety than is included in our present day classifications.

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XVIII.

EPISTAXIS IN PREGNANCY REQUIRING LIGATION OF THE EXTERNAL CAROTID: REVIEW AND CASE REPORT.

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Nosebleed is such a common occurrence that one rarely, if ever, thinks it can be so persistent as to require ligation of the external carotid artery. In fact, two leading rhinologists have said that they have never seen a case of epistaxis that they could not control by packing. This is not surprising, because such a case as will be reported here is extremely rare. However, equally good rhinologists failed to check this one by repeated packings.

Because a nosebleed may vary in severity from a very mild, almost insignificant one to a fatal hemorrhage, a host of remedies and methods have been described and prescribed for controlling epistaxis.

Hays quotes from Sir Morell Mackenzie's book (1884) the constitutional causes of epistaxis:

- "1. Alterations in the constituents of the blood;
- "2. Disease of the blood vessels;
- "3. Obstruction to the circulation causing a strain of the whole system; the nose suffers first because the vessels are superficial and in places the veins are enlarged to cavernous sinuses.
- "4. The blood from the nose may be a vicarious discharge."

Hays adds, "In general, these four causes hold good today and to them may be added: (1) that epistaxis often may be, and is caused by a lowered resistance of the tissues due to various chemical and bacterial toxins; and (2) that epistaxis may result from a pathological change in the nasal mucosa due to a local infection at this site but caused by a general disease." He does not mention pregnancy (perhaps because it is not a disease), nor does he mention the toxemias of pregnancy as a possible cause of epistaxis. DeLee and Williams merely mention the fact that

nosebleed is a common occurrence during pregnancy but do not describe severe cases. As a cause they cite the increased congestion of nasal vessels during pregnancy. Hirst wrote: "The disposition to epistaxis may be so severe as to threaten life. Epistaxis, however, is a more serious complication of parturition than of pregnancy. It can only be checked by the rapid termination of labor. Meanwhile the nose should be packed."

The literature contains only one case (von Broich) in which complete blood studies were made. There is no case in which the external carotid was ligated to control the nasal hemorrhage during pregnancy. In fact, the only case of ligation of external carotid to control so-called essential or uncomplicated epistaxis was reported by Heyde in a man, age 51. In other cases of epistaxis in which ligation of external or common carotid was done the bleeding was postoperative or due to tumor or trauma, or the ligation was performed preoperatively as a prophylactic measure. The literature contains reports of six cases of severe epistaxis during pregnancy, three of which ended fatally.

The first was reported by G. Richelot, in 1847. His patient was a primipara, age 28, who had been troubled with nosebleeds since the age of 7. Her father and twin brother were affected in the same manner from infancy, and her mother had epistaxis at the time of her first pregnancy.

At the age of 13, her color was good but later she was told she suffered from a disease due to poor food. At 19 she had chlorosis. Richelot saw her for the first time in 1839, at age 25, and prescribed iron. She married in 1842 and became pregnant in November. Her condition became more and more serious. Edema increased daily. Labor began on July 29th and on the 30th she gave birth to a living child. Her color improved after delivery and her pulse rate increased from 84 to 108. The patient's condition was serious for twenty days, and then she improved and returned to her previous condition. In 1844, she became pregnant for a second time. Treatment was instituted at once to prevent further hemorrhages and a recurrence of her former condition. Again she was carried through her pregnancy and gave birth to a well developed child.

The second report was of a fatal case of Thomas Hubbard, presented in 1895. His patient was age 40, para XI, within one week of term. Her first severe epistaxis was a year previous and her next was five months before Dr. Hubbard saw her, when she was already four months pregnant.

His first visit was January 10, 1894, when he succeeded in checking the nosebleed by pressure and cautery. Bleeding occurred from the septum. Diagnosis of atrophic rhinitis was made. On January 27th, nine days after a normal delivery of a healthy child, she had another nosebleed, less severe than former ones. This was controlled by cauterization. On February 2nd, her temperature was 103. The next day it was 104. "The lochia and milk were suppressed; lumbar pains continued, and a few days later intense pain in one ankle and right thumb supervened. The condition of the nose remained the same to the end. Death occurred on the tenth day."

Hubbard states: "The occurrence of epistaxis during pregnancy is not rare. I have seen and treated five such cases in which hemorrhage was more or less severe. They were all between the fourth and seventh month. In no case was there any disturbance other than a normal pregnancy, and in only one (just narrated) was there a history of previous severe epistaxis. In all of the cases a ruptured septal artery was located and one cauterization sufficed to cure in four of them."

In 1896, Hutchinson reported an experience that occurred in May, 1871. His patient was a multipara, age 36, in the sixth month of pregnancy. She had had nosebleeds as a girl, and in the sixth month of a former pregnancy she had had a rather severe epistaxis. On this occasion she was in collapse from loss of blood. The anterior nostrils were plugged. He directed that she "be propped up in bed with the head as high as possible, the feet put in hot water, and ice applied to the back of her neck. The bleeding did not return." He states, "epistaxis does sometimes end fatally, especially, I think, in women."

The patient of Von Broich (in 1925) was 31 para III. She, too, had had epistaxis as a child. Present illness: She was having nosebleeds every three and four days, and was seven weeks pregnant. March 21st, three days before admission, the nose was

packed. Hemoglobin was 30 per cent, R. B. C. 2,600,000, W. B. C. 9,700. Diminished platelet count. No young reticulocytes. Slight anisocytosis; moderate poikilocytosis. Bleeding time twenty minutes. Calcium given. March 23rd, sudden severe epistaxis. Nose again tamponed. Pulse smaller and irregular. Fifteen cc. sodium citrate blood given intravenously followed by a chill one-half hour later. Patient died four hours later. Autopsy: Blood in nose, high grade anemia and cloudy swelling of viscera, moderate septic swelling of spleen, tracheobronchitis, nothing characteristic of pernicious anemia, leukemia or scurvy. He concluded that nosebleed was the result of low platelet count because of family history—

One brother had epistaxis in youth.

One sister was a severe bleeder.

One sister, 36 years, unmarried, had nosebleeds and bleeds easily. Her blood was negative.

One sister, 15 years, negative.

Bethel Solomon (1918) reported a patient eight months pregnant with severe epistaxis. This was finally controlled by post-nasal packs but she nearly died. She was delivered of a macerated baby. This patient gave a history of nosebleeds prior to her pregnancy. She left England for India, where she again became pregnant and died from nasal hemorrhage.

With Wilson, Solomon reported another case in 1927. This woman was a primipara, age 27, pregnant twenty-six weeks. She entered the hospital because of a severe epistaxis from both sides. Her blood pressure varied from 200 to 224. Her urine was loaded with albumen. She had edema of her feet. Her pregnancy was interrupted and the epistaxis ceased.

The authors conclude, "If pregnancy is a direct causal factor it is probably on account of increased vascularity of the nasal mucous membrane."

Author's case:

Mrs. A. F., admitted May 1, discharged May 15, 1931, on the service of Dr. Samuel Cowen. Age 27, para II. No previous history of nosebleeds. Family history negative for bleeding. First pregnancy normal in all respects.

P. I.: Sudden onset of epistaxis afternoon of admission to hospital while driving her auto. The patient is three and a half months pregnant. She has been vomiting some for the past two weeks but not feeling ill. Patient is in shock, cold, pulse small, 120, and bleeding from left nostril. Both anterior and posterior nasal packs were inserted; morphin given.

May 2. Condition good. Pulse 110, respiration 14. Pressure 96 systolic. Heart and lungs negative. Abdomen negative except for pregnancy. Blood still oozing through packings. Adrenalin and cocain applied and nose repacked by Dr. Cowen. Bleeding continued all day. Brisk hemorrhage in evening and nose was repacked by Dr. Lieder, 11:15 p. m.

May 3. Oozing of blood continued. At times bleeding was profuse. Patient unable to retain anything in her stomach. Vomitus contains much old and new blood. The bleeding is from the left nostril. Packings removed with considerable loss of blood and new packings inserted by Dr. Cowen at 1:30 a. m., May 4th. Transfusion of 550 cc. whole blood one hour later and infusion of 2,450 cc. 5 per cent glucose given. Total output of urine for twenty-four hours, 150 cc. Urine negative except for high specific gravity.

May 4. Venoclysis of glucose started. Epistaxis continues. Breath foul. Packings changed by Dr. W. Chamberlain. Temperature 100, pulse 120, respiration 20. Urine, 3,900 cc. output.

May 5. Bleeding recurred; packing reinforced and posterior pack tightened. Transfusion, 600 cc. whole blood.

Development of left acute otitis media; spontaneous rupture of tympanic membrane.

May 6. There was a recurrence of bright blood from the left nostril. Ligation of external carotid with two ligatures. Artery cut between ligatures. Operation performed under novocain, block anesthesia, by A. Strauss. Packings were then removed; no bleeding point seen. Region of middle turbinate cauterized with Bovie cautery by Dr. Cowen. Small packing left in.

May 8. No bleeding since the ligation (36 hours). Patient aborted a four to four and a half months old fetus and placenta. About 500 cc. lost. Patient again transfused with 500 cc. whole blood.

May 9. Packing removed. No further bleeding. Purulent discharge from left ear.

May 13. Temperature 98.6, pulse 90, respiration 20. No complaints. Discharged well May 15th.

Urine examinations, May 2nd and May 9th, were negative. Blood counts, May 2nd, W. B. C. 21,900; May 9th, 42,900. R. B. C., May 2nd, 3,220,000, May 9th, 2,550,000; Hgb., May 2nd, 60 per cent; May 9th, 40 per cent. Pm. n., 97 per cent; platelets, 150,000. Clotting time and bleeding time within normal limits.

Pathologic examination of fetus and placenta showed pregnancy approximately four and a half months. Placenta showed areas of degenerative and necrotic changes and early acute inflammation with moderate infiltration with polymorphs. (B. S. Kline.)

The facts noted here agree with the conditions seen in less severe cases of epistaxis in pregnancy, namely, that the bleeding usually occurs in the second half of pregnancy and in women who have had nosebleeds before. Three of the seven cases were primipara and in one the para was not stated.

Unfortunately, the six former cases with one exception were reported without the clinical data we look for today. V. Broich pointed out that Eseh, Rumpf, v. Ottingen, Myer-Ruegg and Clausen described a disease they called anemia of pregnancy resembling pernicious anemia and sought the cause in a toxin of pregnancy causing increased destruction of the red blood cells.

His case had a count of 2,600,000 red cells and a hemoglobin of 30 per cent. The platelets were diminished and the bleeding time was twenty minutes. However, the autopsy showed nothing characteristic of pernicious anemia.

Hubbard's patient died because of sepsis, and it is not clear whether the source was the cauterized area of the nose or the uterus. Nevertheless, the former could have been the cause, as is well shown from the fact that in the writer's case otitis media developed from repeated packing. Other cases at postmortem showed pus in frontal sinuses and mastoids (Jones) and in the nasopharynx and mastoids (Levy) after packings had been used to control nasal hemorrhage in men.

Jones' patient was a colored man, age 25, who had fatal epistaxis following varicella. He concluded: (1) that he was dealing

with "temporary hemophilia"; (2) that packing was useless; (3) that the mastoid and ethmoid infection were secondary to the hemorrhage condition; and (4) that treatment must have for its object to increase the coagulability of the blood." He administered horse serum and thromboplastin.

He does not state the bleeding time or clotting time of the blood nor did he attempt ligation of the external carotid. Solomon and Wilson, believing they were possibly dealing with some disturbance due to pregnancy, decided to interrupt pregnancy to control the nasal hemorrhage. They were successful.

In the author's case no clinical or laboratory examination threw any light on the etiology, and no treatment was effectual until ligation of the external carotid was resorted to. Finally the patient aborted two days later and convalescence was uneventful.

Ligation of the common carotid artery to control hemorrhage was first done successfully by Mr. Flemming, in 1803, according to Bartlett and McKittrick, who reviewed the literature up to 1917, and reported in some detail 105 cases. Seven of these were for spontaneous hemorrhage from the nose or nasopharynx due to tumors or ulcers, etc. The author may add one to this list for a tumor of the parotid that caused a fistula in the cheek and invaded the antrum. Death was caused by metastases.

Ligation of the external carotid alone for epistaxis has been more limited. These were performed as follows: For postoperative nasal hemorrhage: Bartlett and Orr 2, Jackson 2, Cohen 1, Keen 1; for post-traumatic nasal hemorrhage, McKnight 1, Kaufman 1; for prophylaxis preliminary to nasal operation, Jackson 8; spontaneous, Jackson 2. (He does not describe these cases.) Jackson further states that he has ligated the external carotid thirty-eight times without any mortality.

The only case of ligation of the external carotid for uncomplicated epistaxis so far reported is that of F. T. Heyde. His patient was a Russian male, age 51. His first serious nosebleed was sixteen years previous to admission. He had been bleeding one week when first seen by Dr. Heyde. Nasal treatments failed to check the hemorrhage. Ligation of the external carotid was then resorted to successfully.

Author	Yr. published	Previous Epistaxis	Age	Para	Months Pregn't	Treatment	Result	Remarks
Richelot.....	1847	yes	29	I	3 mos.	Local	Well	2nd pregnancy O. K.
Hubbard.....	1894	yes	40	XI	9 mos.	Cantery	Infection Died post-partum	Pyemia
Hutchinson.....	1896	yes	36	multi-para	6 mos.	Local and posture	Well	
Von Broich.....	1925	yes	31	III	7 weeks	Tampon, transfusion, calcium	Died	Suspected pernicious anemia. Autopsy neg.
Solomon.....	1918	yes	not given	not given		Postnasal packs, aborted	Well	Died of epistaxis in following pregnancy
Solomon and Wilson.....	1927	not given	27	I	26 weeks	Induced abortion	Well	Albuminuria
Strauss.....	1932	no	26	II	4 mos.	Local and tampons, 3 transfusions. Ligation external carotid	Well	Aborted 2 days after ligation

Contrary to the high mortality (10 per cent), complications (about 20 per cent) consequent to ligation of the common carotid artery, no death has been attributed to ligation of the external carotid alone. Scudder acknowledges a personal communication from Matas, who said that in 100 ligations of the external carotid he had two deaths, but nothing is given of their histories. Furthermore, it appears that when the hemorrhage has been from the nose only ligation of the external carotid has been successful in every case reported. This is to be expected because the direct blood supply to the nose is through the internal maxillary and sphenopalatine arteries, whereas from the internal carotid it is only indirect through the ophthalmic branch.

Therefore, this review presents three points to be emphasized:

First—Given a case of severe uncomplicated epistaxis when local measures plus transfusion fail, ligation of the external carotid should be performed to save life.

Second—Severe epistaxis during pregnancy may be stopped by terminating pregnancy.

Third—Ligation of the external carotid artery to stop epistaxis in pregnancy should be preferred in order to save the life of the child.

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XIX.

ACUTE MASTOIDITIS: EARLY OPERATION OR DELAYED?

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Much discussion has been devoted to the problem of selecting the best time to operate in acute mastoiditis. A wide divergence of opinion exists as to the merits of early operation, most of the controversy centering around its efficacy in preventing cerebral complications in the fulminant type of case. No hard and fast rule can be adopted that will apply in all cases. Too many factors enter into the picture and too wide differences occur. Signs, symptoms and indications for operation overlap, and types cannot be sharply classified. The type of epidemic changes from one year to the next and sometimes during the same winter. Nevertheless, certain general principles may prove helpful in the average run of cases, but rare exceptions occur and each new case becomes a problem unto itself.

Every case of mastoiditis should, therefore, be studied carefully before the decision is made when to operate. The diagnosis should include a careful estimate of the character and extent of disease probably present in the middle ear, mastoid and surrounding parts. A conscientious effort should be made to visualize what has gone before, what is present, and what is likely to happen. Two definite ideas should be formed and kept vividly in mind: first, what the operation is expected to accomplish and, second, what its chances are for doing so. Many a mastoidectomy has been done on a patient already beyond the aid of surgery and the extra tax on his resisting powers has taken away what little chance he had. Occasionally a mastoidectomy may have to be done desperately, because it appears to be the only avenue of attack open at the moment that offers any hope, but the urgency of many mastoidectomies is more apparent than real.

Some operators apparently believe that a mastoidectomy can always cut off every communication between a tympanic infection

and the brain—if properly timed and skillfully done. Many recognized facts may be cited which tend to refute the soundness of such an opinion.

Direct extension through the mastoid by necrosis, decalcification and destruction of bone is generally accepted as the route to the brain most frequently taken by a tympanic infection. This is the only route the infection can take that will be affected to any great degree by a simple mastoidectomy. An osteothrombotic phlebitis of small veins in the bony partitions between the cells is considered by Eagleton, Kopetzky and others as a short circuiting process of this method of extension. Unless this occurs it should be the slowest method of all if the mastoid were perfectly healthy when the tympanic infection began. In other words, it seems reasonable that a primary or initial attack of mastoiditis will require considerable time to destroy healthy bone and reach the dura in that manner, providing no congenital defects, such as dehiscences, are present. Unhealed traumata should be excluded also, as well as other unrepaired defects left by previous injuries of any kind—bacterial infection, for instance. If the mastoid has been diseased one or more times previously and incompletely repaired or lightly walled off areas of destruction have been left near the dura, one of them might be easily and rapidly lighted up into fresh activity by a new infection and quickly break into the intracranial tissues. Especially is this likely to happen if the last infection has been recent. Then the residual areas of mastoiditis may be incompletely repaired or lightly encapsulated and probably still contain organisms retaining much of their former virulence. It can be easily visualized how important previous infections may be in determining the urgency of mastoidectomy.

Several other factors must be considered in connection with an estimate of the time required for an infection to progress through the mastoid by bone necrosis. Bacteria differ widely in that respect. Streptococci, pneumococcus type III, and the diphtheria bacillus are said to be very rapidly destructive. I had one diphtheritic case and also one of pure colon bacillus infection, each of which apparently was rapidly destructive.

The age of the patient is another factor that may have to be taken into consideration. Kopetzky states: "Except with infants

and young children, operative measures may be postponed with safety longer in the young than in the aged. Osteosclerotic changes are more apt to be found in older than in younger individuals; the thicker and harder the osseous wall enclosing a purulent lesion, the greater the danger of pus spreading in other directions."

The anatomic peculiarities of the mastoid may influence the progress of the disease to some extent. X-ray plates may give anatomic information that demands careful consideration. The position of the lateral sinus may be exceedingly important if it is superficially placed, or if it curves forward markedly so as to crowd upon the antrum. The picture may indicate that the most advanced destruction is very close to the sinus. Classification of the mastoid as to the character of cell that predominates therein may also have to be considered, but I have never been able to estimate it to advantage except that in the eburnated variety the very thing often happens that Kopetzy describes in the aged.

I have seen several cases in which meningitis followed the onset of otitis media so rapidly (without any clinical evidence of mastoiditis) that it seemed reasonable to assume the infection was transmitted by septic emboli from the middle ear. Naturally, that opinion is difficult to prove absolutely, because it is often impossible, even with a thorough autopsy, to trace an intracranial infection to its source. In making such a statement, the fact should not be lost sight of that the absence of clinical manifestations of mastoiditis does not exclude its presence in considerable degree. No one can estimate the exact amount of clinically silent mastoiditis present in a given patient at a certain time. Probably a great deal more mastoiditis accompanies every tympanic infection in its early stages than is usually visualized. It may begin almost simultaneously with the onset of tympanic infection. The mastoid infection often far exceeds the tympanic in severity. The drum and middle ear may be perfectly normal in appearance. In my opinion, a nonsuppurative otitis media always precedes the so-called primary mastoiditis and the same organisms not producing suppuration in the tympanum either take on new virulence in the mastoid or find aid from other organisms already there. When it is known that there are small communications between

the various contiguous mastoid cells, it is easy to visualize a virulent infection quickly reaching a deep cell situated near the dura. This cell may have a thin or deficient wall presenting toward the brain. If any infection arrives there eventually, an extremely virulent one may do so almost at once. These deep cells are naturally the most dangerous and often their involvement is very silent. Frequently mastoiditis is patchy and not at all uniform. The infection may go down into the deeper cells first and not extend widely from them for some time and occasionally not at all. Such mastoids often fail to present many of the usual signs of mastoiditis. Mastoid tenderness will not often be elicited until the cortex and the periosteum are approached by the inflammation. An extremely dense and hard cortex frequently masks what is going on deep beneath the surface, even when the mastoiditis is unusually extensive and destructive. Two cases come to mind, in each of which the mastoid was a mere shell containing pus and bony débris, with the inner table completely destroyed over a large extradural abscess and still no external tenderness, edema or other superficial evidence of inflammation was present at any time. At operation these findings were explained by an exceedingly dense and firm cortex.

Therefore, when the possibilities for extensive but unsuspected mastoid involvement are considered, it must be admitted that any statement regarding the exact route of travel taken by a tympanic infection to the brain in a particular case may be largely a matter of personal opinion (even in the absence of clinical evidence of mastoiditis). An autopsy may give some idea as to which site was the most likely focus, but it does not always settle the matter absolutely.

The route of embolic invasion is thoroughly enough established, even though the exact source of the emboli may not be demonstrable in every case. The ears may not be factors at all in some instances, but they are hard to exclude if involved prior to the onset of an intracranial complication. Otitic brain abscesses have been found far removed from the infected ear, even on the other side of the brain. When they are found, not in close proximity to the affected ear, and the autopsy reveals no probable focus in

the other ear, the nose, throat, lung etc., then the supposition is justifiable that they were produced by septic emboli.

It is possible that the mastoid operation fails in its intended purpose at times, because in spite of the additional drainage afforded backward through the antrum into the wound, considerable infection remains in the middle ear and gets into the blood stream from there after the operation. Overlooked involvement of the petrous pyramid may explain others, and if infection once started along some intracranially leading canal conveying a nerve or blood vessel, eliminating the focus behind by a mastoidectomy might be futile.

If direct extension through the temporal bone occurs rapidly in the middle ear and reaches the labyrinth first, it is doubtful how much the simple mastoid operation alone would aid such a condition, although it might be indicated as the first step in drainage. The assumption that if an infection in the middle ear is severe enough to cause necrosis of the inner tympanic wall, it will also produce considerable coincidental mastoiditis, is certainly justifiable usually. However, I believe that in some instances the mastoid does escape to the extent that it is not of sufficient added menace in itself to require operation. If a suppurative labyrinthitis can be diagnosed, an early mastoidectomy may be urgently indicated, if done in conjunction with a labyrinth operation. Nevertheless, many labyrinth operations have been done too hurriedly. A little patient waiting renders many of them unnecessary. At least, it is doubtful if great haste in such an operation accomplishes the intended result as often as expected. A few symptoms of labyrinthine irritation cannot be interpreted as a suppurative labyrinthitis just because a suppurative otitis media is also present. The tests for a suppurative labyrinthitis must be definite and positive, or mistakes will occur. Even then, some patients probably do better without the added shock and trauma of operation than with it.

At this point it is well to emphasize that this paper is dealing with the problem of when it is best to do or postpone a simple mastoidectomy and takes no consideration of possible future advances in diagnosis or surgical technic when better results will be obtained by doing early mastoidectomies in conjunction with

other surgical attacks upon the complications of mastoiditis and otitis media.

When the foregoing considerations of the various methods of extension of tympanic infections to the brain are reflected upon, it will be seen why the mastoid operation may not always accomplish its intended purpose as a life saving measure regardless of when it is done. The maximum importance of the mastoid operation and when it should be done comes to the fore only when the tympanic infection takes the usual route of extension by contiguity of tissue through the mastoid cells. If there is sufficient evidence of a dangerous spreading process in the mastoid to clearly indicate that a mastoidectomy must be done eventually, then other possible routes of extension may have to be ignored and allowed to take care of themselves temporarily, while the more pressing business at hand in the mastoid is attended to. The first step to take in such a situation is to decide whether to operate at once or to wait. To my mind, this decision chiefly depends upon an estimation of the degree of localization* present in the mastoid at the time the operation is being considered. When is a reasonably safe degree of localization to be found there? This is the crux of the situation, regardless of what the indications for operation apparently are, how virulent is the infection, or how the time element may figure. If good reason exists to believe that there is a well localized process in the mastoid, the operation may be done without delay and it probably will be successful, providing complications have not already set in. If the process within the mastoid is not a localizing affair, an operation probably will not accomplish its purpose and may as well be delayed, even though fatal complications occur in the waiting interval. If they do, it is possible they would have occurred in spite of the operation.

Mild average cases showing no alarming symptoms or indications of extreme virulence should certainly not be rushed to the operating room. It is only the frantic effort to prevent fatal complications in the fulminant type of case that leads to hasty surgery.

*Localization is used here in the sense of circumscribing or walling off and not in the sense of accurate location.

In my hands the early operation has failed to accomplish the desired result in such cases. I have seen distressing results both ways, but I feel sure that on the whole my general average has been better since adopting a fairly conservative course.

A good general rule of surgery is to wait for localization in any infectious or suppurative process if there is a reasonable chance that it will occur eventually. Processes not showing such a tendency at any time are usually poor operative risks. Many of them do better, on the whole, without any operative interference. This is especially true of suppurative conditions capable of unlimited extension throughout any one type of tissue. If an infection becomes implanted in any layer of tissue, containing no effective anatomic barriers to its unlimited extension, the only obstruction to its progress must be an effort at localization on the part of nature.

It must be admitted that exceptions to the general surgical principle of waiting for localization are encountered. Conditions may be selected in which there is a better effort at localization early than later. Brain surgeons maintain that their best chance to save a meningitis patient surgically is lost if they do not get their opportunity early, before the infection becomes too generally distributed. Septic meningitis from ears and nasal sinuses usually begins as a localized affair. Occasionally the localizing effort is successful and it remains so without developing into a general meningitis. The so-called protective meningitis which may show many leucocytes but seldom any bacteria in the spinal fluid, is not very fatal if it does not change its character. If meningitis is developing slowly from organisms reaching the meninges in small numbers at one minute point, the early meningeal infection may be sufficiently localized to be drained if attacked accurately and deftly at the portal of entry by a skilled neurologic surgeon. Waiting might only give time for the infection to break its bonds and become general. Eagleton, Sachs, Klemme and many others plead that the future hope of surgery in meningitis consists in accurately diagnosing the exact portal of entry of the infection and attacking it there early for adequate drainage. This plan sounds feasible enough if a correct diagnosis can be made and a successful drainage operation devised.

Kopetzky described a hemorrhagic type and a coalescent type of acute mastoiditis. If I understand him correctly, he believes

that they are separate and distinct types and not different stages of the same infection. He maintains that the acute hemorrhagic type remains diffuse and bloody to the end, and that if one waits for localization in such a case he will not get it. Doubtless some cases are so. Death occurs too quickly. However, they surely do not all die within the first few days. If they do not all die early, unless operated upon, what becomes of them later? How do they repair if nature does not localize, wall off and heal them? I have never seen a case in the fifth or sixth week of infection except when good and sufficient reason existed to believe that an acute exacerbation, due to a new infection, had recently occurred.

Assuming that Kopetzy is right and that cases do occur which will not localize eventually, if given the opportunity, the question is still not definitely answered as to whether they do any better by early surgery than delayed. If they are diffuse and at all comparable to other diffuse, noncircumscribing inflammations, they should never become good surgical risks. Since adopting the more conservative waiting policy I have seen several of these cases, giving every indication known to me of extreme virulency, quiet down and turn out to be not so desperate as they first appeared. Some of them eventually came through without operation at all, while I was waiting for better localization. Not being as virulent as they first appeared, perhaps they would have stood operation well and have been catalogued as potential meningitis cases when they really were not.

Aside from the standpoint of the prevention of otitic meningitis and other usually fatal complications of acute mastoiditis by early or late operation, there is also the question of which procedure is followed by the better convalescence and postoperative result in the various types of mastoiditis. There seems to be fairly general agreement that early operation is followed by a longer and a more stormy convalescence and slower healing of the wound in almost every type of infection. As a general rule, such a course results in more severe constitutional reactions and higher postoperative temperature. The wounds drain longer and do not unite as well. However, if the patient's life is saved, and he gets well eventually, that is of chief importance. The character of his convalescence is soon forgotten.

Early operation is not a definite term. I have seldom been able to estimate accurately in hours, days or weeks just what others meant by "early." I have read statements that an operation was performed on the fifth day, we will say, but still have been unable to find whether it was the fifth day after the onset of otitis media, after the onset of mastoiditis or something else. If any standardization of this angle of the subject exists in the literature or in the minds of the profession at large, I am unaware of it.

There should be some recognized starting point from which to reckon time so that all would be speaking in the same terms. It is most exasperating to read a case report in which the time of successive events cannot be figured accurately. At least, a writer on the subject should make it clear to his readers how he calculates the time element when he speaks of an early or late operation.

To my mind, the only dependable starting point possible is the onset of otitis media as evidenced by the hour of onset of earache. The first clinical manifestation of the onset of mastoiditis does not fix that hour as the true onset of this complication. That has already been discussed sufficiently in this paper. The reverse usually is true of the onset of a tympanic infection. The tympanic membrane especially is extremely sensitive. It usually telegraphs to the brain in unmistakable terms of pain promptly whenever any traumatic, chemic, toxic or bacterial injury is sustained. Therefore, the onset of earache is usually a fairly reliable guide to the onset of otitis media. Often it precedes any otoscopic evidence of tympanic inflammation, and otitis media does not go very long before it can be detected with an otoscope. Mastoiditis goes on longer without being detected, and a mastoid may be opened within the first twenty-four hours of the appearance of clinical symptoms in the fourth or fifth week after the onset of otitis media and still prove to have been operated on very late. Therefore, we can reckon the possibilities in age of both processes much more accurately from the onset of earache.

In my opinion, a mastoidectomy done within one week, or less, after the onset of earache is an extremely early operation. One week after clinical evidence of mastoiditis first appears is usually early, but there are more exceptions to this statement than to

the former. I always feel ill at ease when I find it necessary to open a mastoid before the third week after the onset of earache. Patients do better if they can be nursed along until the beginning of the fourth week or later. I will plead guilty to having violated every rule that I have ever been able to formulate on the problem of early or late operation. Some case always comes along that presents certain features which seem to demand that it be handled differently from others of its general class. The time element figures only when it definitely suggests that the condition apparently observed in the mastoid could not be present for lack of time for it to form.

Several sound principles can be outlined which have important bearing on the decision of when to operate in certain types of acute mastoiditis.

One of the most useful of these, in my experience, has been an appreciation of the difference in the destruction probably present in the mastoid during a primary or initial attack of mastoiditis at a given time after onset compared with what may be found at relatively the same time during a second, third or any subsequent attack. Kopetzky has most admirably emphasized the increased danger of delay with each subsequent attack. Others have brought out the same point. It has been noted repeatedly that the urgency of early surgery in many cases is explained at operation by finding an old, dormant process dangerously lighted up by a new infection. Early operation is increasingly more justifiable with each recurrence. Especially may an early mastoidectomy be urgently indicated if the last infection has been recent. The reasons for these opinions have already been gone into sufficiently in this paper. Some might suggest that the danger of a second or subsequent infection could be figured two ways, either of which might prove correct. This is true. One might choose to estimate that instead of being weakened by the previous infection, the mastoid probably would establish a certain degree of immunity to infection that a virgin mastoid might lack. He might further suggest that the old pathologic process may become so well healed and blotted out by dense callus and new bone that the progress of a subsequent infection would meet more, rather than less, resistance. In certain selected cases this latter suggestion might

prove correct, especially if a long and healthy period of time had elapsed between attacks. However, such a position is a dangerous one to take, because it is against the law of averages. Experience teaches that just the opposite is what usually happens. If not perfectly sure of such an opinion (and how can one be sure?), it would be better to operate. In my experience, a second or subsequent attack of mastoiditis stands operative trauma well and early operation better than a primary mastoiditis usually does. Single X-ray plates are often worse than useless in determining the exact degree of pathologic change present in the mastoid. They may be positively misleading. They do not always differentiate between old and new destruction. Only comparative plates, taken several days apart, are accurate in showing the progress of a new infection. When this information is needed most, time is the important factor—i. e., in deciding whether or not to operate at once. Therefore, the time is usually not available to wait for repeated pictures, as an immediate decision is imperative. Neither do blood counts and other laboratory procedures always give the desired information. Therefore, one must fall back on instinct, judgment, clinical experience and the law of averages.

Another observation of great importance, in my opinion, is the duration of time between the onset of earache and the onset of clinical evidence of mastoiditis or other complications of otitis media. This interval is often a reliable indication of the virulence of the infection when no other distinctive signs or symptoms may be present. The shorter the interval, the more virulent the infection. I also believe the history as to previous ear infections determines how cases should be handled when this interval is short. If previous ear inflammations have occurred, early operation may be urgently indicated or is at least justifiable. If the attack under observation is an initial or primary one, early operation will probably do no good and frequently actual harm.

BEAUMONT BLDG.

The Cause of Otosclerosis.*

(CONTINUED.)

THE PHYLOGENESIS OF THE EAR.

LOUIS K. GUGGENHEIM, M. D.,

ST. LOUIS.

We begin our study of the phylogensis of the ear with the lowest form of vertebrate life,¹ the *cyclostomata* (round-mouths). According to Brehms,² these vertebrates have elongated, worm-like bodies of about the same circumference throughout. There are but few scales on the thick, slimy skin. The skeleton is of pure cartilage and very incomplete. The spinal column consists chiefly of the chorda, which is surrounded by connective tissue and areas of unconnected cartilage. The skull is of cartilage plates separated by membranes; a very incomplete brain capsule. The special sense organs, only, are protected by firm cartilage. Branchial arches are absent. The teeth consist of hornified areas in the mucosa. The alimentary tube is not divided into stomach, intestine, etc. A liver is present. According to Maximow,³ the spleen is found in all verterbrates except the cyclostomes.

The nervous system is poorly developed, the brain small and poor in ganglion cells. The aural apparatus is of very low development. The olfactory organ has a single outer opening which leads to a nasal capsule supplied with two nerves. There are two large eyes and a third or vertex eye. From the midbrain a nerve passes to the cranial roof and at its end shows a vesicle, on the floor of which is a layer of cells similar to those of the retina. The roof of the vesicle consists of transparent cells (lens). Above the vesicle the cranial roof is transparent. This is phylogenetically a very old organ. The heart, vessels, kidneys and genital glands are similar to those of fishes.

*From the Oscar Johnson Institute of Washington University.

The *cyclostomata* have very powerful tongues. They enter the branchial clefts of fishes or bore into the soft parts and devour their prey.

Cyclostomata may be divided into two classes: (1) The *myxini-dae* (Gr. myxa-slime), including the typical hagfish, and (2) the *petromyzontidae* (eel-like parasitical fish), including the typical lamprey.

The *myxinidae* comprise the lowest of all vertebrates. The myxine glutinosa L. (the hag) (Fig. 7-A) is common in the North Atlantic. Its length is about 50 cm. Its color is bluish-white. Both ova and spermatazoen are found in their bodies. Whether in adult life both sexual glands function is not known.

The *petromyzontidae* have a stronger cartilaginous skeleton than the *myxinidae*. They swim poorly, so get about by sucking onto some fish. Like the hag, the lamprey gets its nourishment by boring into fishes and eating the soft parts. When they rid themselves of ova or spermatazoen, the parents die.

The *petromyzon fluviatilis* (river-lamprey) (Fig. 7-B) is the best known of the family. It is about 50 cm. long, weighs 100 g., is of a glistening greenish-blue color, sides of light yellow. The belly is silver white, the fins violet.

According to Anders Retzius,⁴ the labyrinth cartilage of the myxine glutinosa (hag) comprises a pair of brownish vesicles (Fig. 1-A) attached to the cartilaginous frame of the head. The upper inner sides of the vesicles are open except for a fibrous membrane. The capsules are oval, show within, a cartilaginous column supporting the membranous aural labyrinth (Fig. 1, B and C.)

Johannes Müller⁵ found that the aural labyrinth of the myxine glutinosa consisted, as did the base of the skull, of bone and cartilage. The capsules were found closed except on their upper surface, where there was found elliptical openings covered with a fibrous membrane through which passed the auditory nerves. The outer surface of the capsule was convex, the inner flat. The capsules were lined with a delicate membrane, within which rested the membranous labyrinth: the latter was a simple ring-shaped canal. The superior portion of the ring was thicker, showing two areas representing the endorgan of the nervus acusticus. This thickened area Müller looked upon as utriculus; the remainder

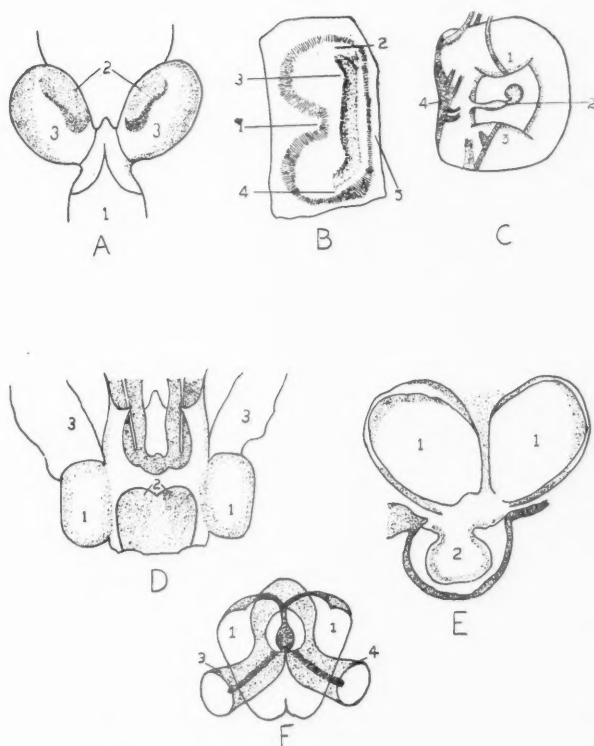


FIGURE 1.

A. Otic capsules of *Myxine glutinosa*, L. 1. Chorda dorsalis. 2. Median openings covered with membranes. 3. Cartilaginous capsules.

B. Membranous labyrinth of *Myxine glutinosa*, L. 1. Perilymphatic space. 2. Ampulla anterior. 3. Otolith membrane. 4. Ampulla posterior. 5. Cartilaginous capsule.

C. *Myxine glutinosa*, L. 1. Crista communis. 2. Ductus endolymphaticus. 3. Crista ac. 4. Macula acustica communis.

D. Otic capsule of *Petromyzon fluviatilis*, L. 1. Cartilaginous otic capsule. 2. Brain cavity. 3. Cranial cartilage.

E. Membranous labyrinth of *Petromyzon fluviatilis* L. 1. Vestibulum (utricle). 2. Sac-like process (sacculus).

F. Membranous labyrinth of *Petromyzon fluviatilis*, L. 1. Vestibulum (utricle). 2. Sac-like process (sacculus). 3. Posterior semicircular canal. 4. Anterior semicircular canal.

—After Gustaf Retzius.

as a semicircular canal. No sign of an otolith was found. The nervus acusticus was just behind the nervus facialis and in front of the vagus.

Ibsen,⁶ on the other hand, found a "saccus lapilli cylindraceus, intima in parte otoconiorum fixorum stratura praeditus," a ductus semicircularis externus and two "ampullae ductum semicircularem cum sacco lapilli copulantes, ambae nervulis ornatae."

According to Gustaf Retzius,¹ the ear capsules of the myxine glutinosa are oval and located on either side of the medulla. On the inner side of each is an opening covered with a fibrous membrane through which pass nerves to the labyrinth. There is no lateral opening corresponding to oval or round window.

Weber⁷ states that the petromyzon differ from other fish by the absence of well developed semicircular canals and otoliths, and also in that the auditory organ consists of a single chamber, a vestibule (utricle). The elliptical cartilaginous capsule (Fig. 1-D) shows toward the intracranium two openings (Fig. 2-A), a small upper one for a vessel and a larger lower one, closed by a membrane which is perforated by the nervus acusticus. The nervus acusticus passes to the membranous vestibulum (Fig. 1-E), spreading out on its walls. He found the vestibulum divided into a lower and an upper cell; the upper one the larger. Rudimentary semicircular canals (Fig. 1-F) were seen. An aquaeductus vestibuli passed above the nervus acusticus to the intracranium.

Müller⁸ found that the true structure of the petromyzon labyrinth was hidden by a membrane between capsule and membranous labyrinth. Within, a sac was found which showed grooves on the upper and lower walls. These grooves each formed two symmetric cells. Over the cells lay two semicircular canals which entered the vestibulum. Both canals showed ampullae. On the floor of the vestibulum was an opening communicating with a diverticulum, smaller than an ampulla. No calcium deposits were found.

Ecker⁹ saw, in the semicircular canals, cilia with lively movements. A sac-like diverticulum of the vestibulum was looked upon as a representative of the cochlea. The capsules were of hyalin cartilage covered with perichondrium and lined with an endothelial membrane.

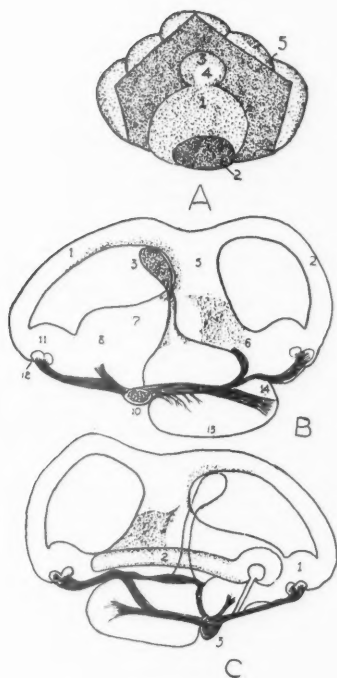


FIGURE 2.

A. *Petromyzon fluviatilis*, Median wall of capsule. 1. Dura mater covering large opening in cartilaginous capsule. 2. Acusticus. 3. Upper opening covered with membrane. 4. Opening in smaller membrane. 5. Cartilaginous capsule.

B. *Acipenser sturio*, L. Median view of membranous labyrinth. 1. Canalis m. anterior. 2. Canalis m. posterior. 3. Saccus endolymphaticus. 4. Ductus endolymphaticus. 5. Sinus utriculus superior. 6. Canalis m. externus. 7. Utriculus. 8. Ampulla externa. 10. Acusticus. 11. Ampulla anterior. 12. Crista ac. amp. 13. Sacculus. 14. Lagena cochleae.

C. *Acipenser sturio*, L. External view of membranous labyrinth. 1. Ampulla canalis m. externus. 2. Canalis m. externus. 3. Acusticus.

—After Gustaf Retzius.

The membranous labyrinth filled the capsule except for a narrow perilymphatic space, and comprised an oval vesicle (vestibulum-utriculus) about 3 mm. in length, 2 mm. wide and 2 mm. high, a sac-formed process (sacculus), two semicircular canals, a commissure, two ampullæ and the nervus acusticus with its branches. No aquaeductus cochleæ was present, but there was an aquaeductus vestibuli. A long macula acustica represented the combined macula rec. utriculi and macula sacculi. No lateral opening, such as oval or round window, was present.

PISCES.

In the spinal column of fishes is always found a very large chorda dorsalis, which in the human disappears before birth.

Fishes are divided into two great classes: (1) cartilage fish (*chondrichthyes*), including the sharks, the rays and the sea-cats; (2) bone fish (*osteichthyes*), including all other varieties.

In the development of the cranium in bone-fish there is formed first, cartilage. When bone appears the cartilage is more or less destroyed (areas of cartilage remaining in all fish).

In the aural capsules a number of ossification centers appear, the most important of which is the prooticum or petrous portion. In addition we find the postfrontale or sphenoticum and the squamosum or pteroticum.

The nervous system comprises the spinal cord with the spinal nerves and the brain. The brain is poorly developed but does show the typical five divisions. The fore-brain shows the hemispheres, poor in nerve cells. The hind-brain holds the most important cranial nerves. The cerebellum is concerned with equilibrium. Anteriorly is the olfactory lobe, highly developed, particularly in the shark.

The brain does not nearly fill the cranial cavity, being surrounded by a semifluid fatty mass.

The organ of equilibrium is composed of the powerfully developed semicircular canals, utriculus, sacculus, lagena and the cerebellum. A true organ of hearing probably does not exist. Only in the dwarf-sheat fish (*silure*) and the amiurus has hearing been definitely proven to exist.

Some fishes are able to breathe atmospheric air. Lung-fish are so called because their swim-bladder is utilized as their breathing

organ. In such cases the swim-bladder is traversed by septa containing blood vessels. The swim-bladder, which in its position corresponds to the lungs, has as its chief function the regulation of suspension in water. By changing its contents, the fish is enabled to change its weight and thereby raise or lower its body without swimming. The filling may be with air or oxygen.

In certain fishes (cypriniformes) the swim-bladder is connected with the internal ears by means of evaginations of the bladder or through a series of four small bones (Weber apparatus). This arrangement functions as a barometer indicating changes in water pressure as the tension of the swim-bladder changes. The four bones united by soft tissue are held in position somewhat like the ossicles, between drum membrane and labyrinth capsule, and transmit pressure changes from the swim-bladder to the labyrinth.

GANOIDEI.

This division includes the sturgeons, bowfins, etc. (Gr. ganos—brightness.) The scales have an enamel-like appearance. As an example of this division, we shall consider the *acipenser sturio*, L. (Fig. 7-C), or common sturgeon.

Koelreuter¹⁰ describes the aural apparatus of the sturgeon and mentions the nonexistence of outer openings. Three branches of the acusticus are traced to the three ampullæ.

Monro¹¹ found, in the sturgeon, three semicircular canals (Fig. 2-C-D), in wide cartilaginous passages; an anterior and posterior vertical and a middle (horizontal). Each canal showed a bulbous end. The anterior vertical and horizontal joined anteriorly. No external auditory canal was seen.

Breschet¹² found no outer avenues of communication with the labyrinth. On the posterior inferior part of the sacculus was seen a small bone (Fig. 3-A), which was fixed between sacculus and cartilage wall. He considered this bone the homologue of the stapes (or of all ossicles) of higher vertebrates.

Retzius¹³ found that the membranous labyrinth consisted of utriculus with sinus utriculi superior, recessus utriculi, sacculus with lagena cochleæ, ductus endolymphaticus, canalis m. anterior, exterior and posterior. The nerve endings were macula acustica, recessus utriculi, macula acustica sacculi, papilla acustica lagenæ

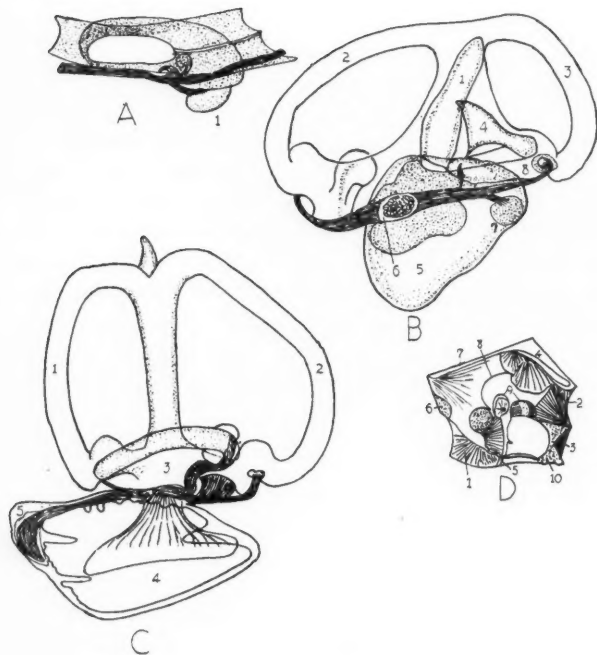


FIGURE 3.

- A. *Acipenser sturio*, L. 1. Bone of Breschet.
 B. *Lepidosteus osseus*, L., and *Amia calva*, L. 1. Ductus endolymphaticus. 2. Anterior canal. 3. Posterior canal. 4. Utriculus. 5. Sacculus. 6. Acusticus. 7. Lagena cochleae. 8. External semicircular canal.
 C. *Perca fluviatilis*, L. 1. Posterior canal. 2. Anterior canal. 3. Utriculus. 4. Sacculus. 5. Lagena cochleae.
 D. *Perca fluviatilis*. Aural capsule as seen from the median side (membranous labyrinth removed). 1. Prooticum. 2. Occipitale laterale. 3. Occipitale basilare. 4. Occipitale superius. 5. Parasphenoidale. 6. Alisphenoidale. 7. Parietale. 8. Anterior canal. 9. Ostia canalis externi. 10. Fovea sacculi et lagenae.

—After Gustaf Retzius.

cochleæ, macula neglecta and three cristæ. On the posterior surface of the sacculus was found a small oval bone near the papilla lagenæ and attached to the cartilage wall of the capsule. Retzius could not say whether or not this bone was the homologue of the ossicles of man.

Lepidosteus osseus L. (Alligator Fish) (Fig. 7-D).—One to one and one-half m. long, olive green in color, glistening silver belly. It comes to the surface to breathe in hot weather.

Retzius found the membranous labyrinth in a capsule with an inner opening covered by dura. The same membranous labyrinthine parts (Fig. 3-B) were found as in the sturgeon. The utriculus, sacculus and lagena all showed an otolith. No mention is made of an outer opening (oval or round window).

Amia Calva L.—Sixty cm. long (Fig. 7-E), of dark olive-green color, belly bright yellow. Labyrinth capsule is of cartilage and bone and shows the usual medial opening covered with dura. No mention of oval or round window has been found in the literature.

TELEOSTOMI (GENUINE BONE-FISH).

This order includes the majority of all living fish. Of 12,000 forms of fish life, 11,500 belong to this class. They all show in common complete ossification of the skeleton. Very important is the fine differentiation of the skull bone and complete ossification of the vertebræ.

The first complete description of the auditory apparatus of the teleostomi was by Ernst Heinrich Weber.¹⁴

The membranous labyrinth lies in the intracranial cavity without a separating membrane and is bathed in cerebrospinal fluid. Weber described the vestibulum (utriculus), saccus (sacculus) and three canals.

In *cyprinen*, *silurus* and *cobitis* the membranous labyrinth is connected with the air-bladder by means of a sinus impar. Three bones of the ear articulate with the first three vertebræ. These bones Weber named stapes, incus and malleus. He noted posteriorly openings which he considered the fenestræ vestibuli ossei.

In certain other fishes of this group there were no ossicles, but canals passed from the air-bladder to the skull and connected up with the auditory apparatus.

Retzius presents in his studies of bone fishes *Perca fluviatilis* L. This is the common European perch. The aural apparatus (Fig. 3-C) lies on either side of the brain, separated from the latter by dura and surrounded by perilymphatic tissue rich in fat. The aural capsule consists of cartilage and bone. The bones which cover the ear apparatus are occipitale basilare (Fig. 3-D), occipitale laterale, occipitale superius, epoticum, prooticum and squamosum. It was questionable to Retzius whether or not one could speak of an aural capsule as in man.

Clupea harengus L.—In the herring, Weber noted that the two membranous labyrinths were joined by a transverse membranous canal, passing under the brain, and from one utricle to the other. He could pass fluid from one to the other.

Breschet found an upper as well as a lower tubular connection between the ears so that a veritable ring around the brain connected the two aural structures.

Haase¹⁵ found the two connecting canals also and in addition that the aquaeducti vestibulorum also joined.

Weber, Breschet and Haase mention a vesicular air containing room (tympanum?) on either side of the skull. These air sacs were in connection, through small canals, with the anterior ends of the air-bladders. They also described the "first homologue of the foramen ovale which comes into existence through the fact that the prooticum, opisthoticum and occipitale basilare do not join closely inferiorly but leave an opening which is covered by a delicate membrane (mucosa of the pharynx).

Esox Lucius L.—The pike's labyrinthine capsule (Fig. 4-B-C) is formed by the occipitale basilare, occipitale superius, occipitale laterale, prooticum, epioticum and squamosum. These bones are joined by cartilage. This is the first vertebrate to show an appendix utriculi (Fig. 4-A).

Anguilla Vulgaris Turt.—In eels the aural labyrinth is small; the bones forming its capsule are joined by cranial sutures and show only traces of cartilage.

Ostracion cornutus.—The "Kiefferfisch" is of interest in that it seems to have no ductus endolymphaticus.

The *Tetrodon mappa*, Less, likewise, has no ductus endolymphaticus.

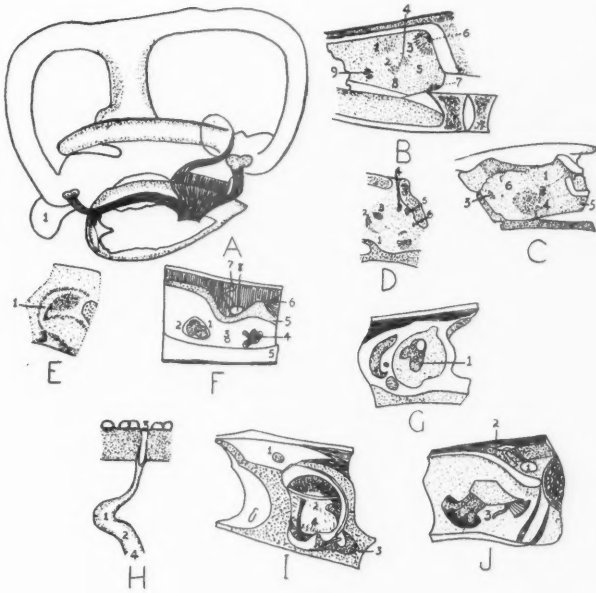


FIGURE 4.

- A. *Esox lucius*, L. 1. Appendix utriculi.
 B. *Esox lucius*, L. Cartilago-osseous otic capsule (external wall).
 1. Fovea ampullaris anterioris. 2. Fovea recessus utriculi. 3. Prototicum. 4. Ostium canalis externi. 5. Cartilage. 6. Ostium canalis posterioris. 7. Canalis vagi. 8. Fovea sacculi et lagenae. 9. Canalis trigemini.
 C. *Esox lucius*, L. Floor of otic capsule. 1. Canalis externus osseus. 2. Cartilage. 3. Canalis trigemini. 4. Fovea sacculi et lagenae. 5. Canalis vagi. 6. Fovea recessus utriculi.
 D. *Chimaera monstrosa*, L. External wall of otic capsule from within. 1. Fovea recessus utriculi. 2. Fovea ampullaris ant. 3. Fovea ampullaris ext. 4. Canalis ant. 5. Canalis post. 6. Canalis ext.
 E. *Chimaera monstrosa*, L. External capsule wall from without.
 F. *Acanthias vulgaris*, Risso. Median view of external wall of otic capsule. 1. Acusticus. 2. Trigemini. 3. Glossopharyngeus. 4. Vagus. 5. Cartilage of skull. 6. Body muscle. 7. Skin. 8. Oval window of Scarpa covered with membrane.
 G. *Acanthias vulgaris*, Risso. Median wall of otic capsule, outer surface. 1. Grooved passage leading externally to the oval opening.
 H. *Scyllium canicula*, Cuv., and *Squatina angelus*. 1. Saccus endolymphaticus. 2. Ductus endolymphaticus. 3. Apertura duct. endolym. ext. 4. Sacculus.
 I. *Raja clavata*, L. Opened capsule from the median side. 1. Oval window. 2. Canalis memb. ext. 3. Lagna cochleae. 4. Sacculus.
 J. *Raja clavata*, L. Median view of external capsule wall. 1. Oval window. 2. Ductus endolymphaticus. 3. Medulla oblongata.

—After Gustaf Retzius.

Siphonostoma typhle Kp. possesses a ductus endolymphaticus. Between sacculus and lagena is a small oval opening representing the beginning of the canalis sacculo-lagenaris (re-uniens).

Hippocampus brevirostris, Leach.—In the short-snouted sea-horse the utriculus and sacculus are combined into one large cavity with which very rudimentary semicircular canals and lagena communicate. Despite these deviations from the usual, the nerve endings are all quite typically present.

Elasmobranchii (Gr., elasmos-metal plate + L. branchia-gills).—Vertebrates having no membrane bones to the skull or external girdle, claspers to the ventral fins of males, five (rarely six or seven) pairs of lamelliform gills, complicated brain with optic nerves forming a chiasma.

Chimaera monstrosa L.—A genus of selachians called sea-rat, king-fish, etc., 1 m. long with a rat-like tail. Color gold, brown and white. The aural labyrinths communicate through small canals (endolymphatic ducts) with the surface, where both canals open externally in one common fenestrum. No vestibular window is present. The external wall is pictured in Fig. 4-D and E.

Plagiostomen.—The shark (*Selachioidei*) is a cartilaginous fish, with transverse mouth. The larger varieties give birth to living young; the smaller variety lay eggs.

According to Monro,¹⁶ the shark shows external auditory meati which lie on the upper posterior portion of the head. Conchæ communicate through small cylindric passageways with large sacs (vestibula) filled with a mucoid fluid and a soft, chalky substance. With the vestibulum (utriculus) communicate three canals.

Scarpa¹⁷ reported, in the shark, an oval window covered with a tense membrane.

Cuvier¹⁸ states that the vestibulum (utriculus) of the shark is triangular. One angle elongates into a canal which passes through the skull to the outside, where it is covered with a thin membrane. The canal opening corresponds to the oval window; the membrane covering this "oval window" functions also as a drum membrane.

Acanthius vulgaris, Risso, is the commonest example of the thorn or spine-shark (*Spinacidae*). Length, 1 m.

Retzius describes the cranium as cartilaginous with a cartilaginous separation of the membranous labyrinth from the brain

(instead of the usual membrane). The capsule (Fig. 4-F-G) is composed of one continuous plate of cartilage. The roof of the capsule shows two openings, one covered by a membrane. On the inner wall of the vestibulum is a groove which passes upward and through the cranium to an outer oval opening, covered by a membrane. Another smaller opening on the surface is the outlet of the ductus endolymphaticus. The capsule is of hyaline cartilage.

Scyllium canicula, Cuv.—The cat-sharks (*Scyllidae*) belong to the cartilage fishes which lay eggs. They have five branchial openings. The *scyllium canicula*, large spotted cat-shark, is 50 to 70 cm. in length; color, red with brown spots, belly white. The otic capsule is of cartilage. The ductus endolymphaticus (Fig. 4-H) opens on the surface.

Squatina angeles. Although a typical shark is very different from a typical ray, there is a transitional form, the angel-fish (*Squatinidae*). The *squatina angeles* has an otic capsule of cartilage. The posterior canal communicates directly with the sacculus. The lagena cochleæ is a small sac attached to the posterior portion of the sacculus.

Ray (Batoidei), fish with flat bodies, of forty different varieties, some having remarkable electrical organs (Brehms).

Camper¹⁹ found in the ray a complete capsule of cartilage. No external auditory canal was discovered. This fish is the first to present a capsule entirely independent of the cranium.

Geoffroy²⁰ found in the cartilaginous capsule of the ray an oval opening posterosuperiorly, which was covered with a membrane.

Monro²¹ describes, in the ray, in the posterior part of the head, two pinhead sized openings, each of which led to a wide curved canal. The two canals were divided by a thin septum. Each canal ended in a funnel from which a narrow canal passed on. These are the external auditory canals. No drum membranes were found. The canals opened into a cavity containing a white opaque mass and a watery, sticky fluid. The canals also contained such a fluid. The cavity contained the membranous labyrinth.

Scarpa²² found superoposteriorly, on the head of the ray, a depression in which were two tense little membranes like drum membranes. These covered two oval windows. Upon removing the membranes one entered the vestibula. In each vestibulum lay

the stone sacs. Around the sacs was a cellular network and water. Three stone sacs were found, all of a different size. The largest, shaped like an almond, lay against the outer vestibular wall, just next the oval window. There were three canals. The membranous vestibulum comprised three divisions: the middle and largest showed a canal which passed upward and through the oval window. Anteriorly lay the second division, which communicated with the middle division. Posteriorly the smallest division or appendix also communicated with the middle division. Two chalk-like gelatinous bodies were found: one in the middle and posterior division, one in the anterior division.

Raja clavata L.—The thorn-ray is $1\frac{1}{2}$ m. long, 1 m. wide. In southern waters it is 3 to 4 m. long and 2 to 3 m. wide.

Retzius found in the thorn-ray that the membranous labyrinth is closed off from the brain by a complete cartilaginous capsule of one piece. In the posterosuperior portion of the capsule is an oval opening covered by a fibrous membrane. This was named by Scarpa, fenestra ovalis.

The capsule (Fig. 4-I-J) is of hyaline cartilage covered by a layer of calcareous incrustation.

Dipnoi.—A class of fishes with regular gills, a single or double lung, nostrils inside as well as outside the mouth. Called lung-fishes. The lung consists of one or two membranous sacs which lie above the gut and open through a glottic cleft into the alimentary canal. These fish come to the surface to breathe air. (Brehms.)

Ceratodus, *Forsteri*-Krefft, a large dipnoan mud-fish, called also *neoceratodus forsteri* and *barramunda*. Found in Australia.

Günther²³ found the ears completely surrounded by cartilage without openings except for nerves. The closed off large recessus utriculi from the utriculus proper and the communication of the anterior and outer ampullæ with the sacculus speak for an intimate relationship, phylogenetically, between the ears of these fishes and those of the *elasmobranchii*, particularly the *chinaera*.

Protopterus annectens, *Owen*, called salamander-fish. During the dry season this fish covers itself with mud except for a small opening through which it breathes (exclusively with lungs).

Bischoff²⁴ writes that the *Lepidosiren paradoxa*, an eel-like lung-fish, closely related to the *Protopterus annectens*, shows no external auditory canal, no oval window and no ossicles.

Owen,²⁵ in a monograph dealing with the *lepidosiren annectens*, states that the aural organ has a thick cartilaginous capsule without any outer communication other than the openings for nerves. There is no sign of a tympanum or eustachian tube, cochlea, round window or oval window.

Kingsley²⁶ writes that recent experiments tend to show that in the fishes the ears are without auditory function and are solely organs of equilibration.

Tait,²⁷ in a most interesting article, "Is All Hearing Cochlear?" states that the cochlea is purely a mammalian organ, fishes, amphibians and reptiles having neither cochlea nor organ of Corti. Instead they have a lagena. The semicircular canals and utriculus of all vertebrates have as their function the control of equilibrium. The function of the sacculus is as yet not certain. A number of investigators have failed to establish a connection between the sacculus and posture.

Of the two functions of the inner ear, audition and equilibration, in the former the eighth nerve subserves an exteroceptive function; in the latter, a proprioceptive rôle. Next the author explains how the two functions became associated. Originally the ear served solely as a proprioceptive organ. Through differentiation it acquired auditory attributes. In the crustacean, *Mysis*, there are a pair of otocysts in the terminal segment of the tail. Removal of these sense organs gives rise to: (1) disorientation (no longer swims back-up); (2) when placed in a glass aquarium, each time the glass is tapped with the finger the animal makes a sudden leap in the water; this reflex fails when the otocysts are removed. One and the same otolithic organ serves the double function of proprioceptor and exteroceptor. The sensing of the water tremor is essentially auditory.

Sound in physics means not what is commonly understood by the word, but the transmission of all molecular, elastic vibrations through gases, liquids and solids. Almost every event on the surface of the globe sets up a series of molecular oscillations. The cochlea is sensitive only to vibrations in the air. Not only are

animals, in the interest of self-protection, sensitive to externally arising tremor, but many of them use this sense for purposes of racial preservation; they take advantage of the physical property of solid and liquid bodies as well as air by which tremors are readily conveyed from their origin to a distance. For the purpose of attracting distant partners, animals devised means of themselves giving origin to propagated tremor. This finally gave rise to articulate speech.

Tait mentions first the croak of the frog which he designates a vocal signal. It is doubtful if the vibrations reach the female through the air. The croak of the frog sends out in water an easily perceived tremor. With the hydrophone this tremor is audible three meters away. In croaking the frog emits no air from the anterior respiratory passages. There is an instrument, invented by Dr. Louis V. King, which transmits tremors in water for twenty miles.

Some fishes when removed from the water emit audible sounds. The sounds are produced by the air bladder. The air bladder acts as a hydrostatic organ, and in some species as a tremor emitting apparatus. In the latter the air bladder consists of two compartments separated by a constriction. The posterior compartment communicates with the pharynx by means of a duct. A chain of ossicles (Weberian ossicles) connects the front of the anterior compartment with the labyrinth. By means of muscles, the air in the anterior compartment can either be thrown directly into vibration or be made to vibrate by being forced through the constriction that separates the compartments. All members of this group communicate with each other by means of the air bladder apparatus. The air bladder that emits the vibration is adapted to receive and resonate to the particular note when externally produced. In this way the members of a particular species are attuned to each other but to no other.

E. H. Weber, who first carefully described the chain of ossicles, believed they subserved an auditory function. He called the three ossicles malleus, incus and stapes.

An American siluroid, *Amiurus nebulosus*, responds to whistling in the vicinity of its aquarium.

Parts of the body other than the inner ear are provided with tremor receptors. Tait hazards the guess that the Pacinian corpuscles are extralabyrinthine receptors. In delabyrinthized rattlesnakes there occurs a rattle in response to tremor stimulation. The tremor receptors lie not in the labyrinth but in the middle three-fifths of the ventral part of the body, the part used by the alert animal for maintaining contact with the ground. The posterior or nonequilibrium part of the labyrinth of lower vertebrates contains four different receptor organs: the sacculus, the lagena, the pars neglecta and the pars basilaris. For the last two, no one has offered a suggestion as to function.

Piper found that the ear of the pike (*Esox*) is responsive to water-conveyed sound. Ross proved that the lagenar and saccular maculae of fishes are concerned with sound reception.

Tait states his provisional conclusion concerning the function of the saccular macula in mammals and in man is as follows: The cochlea with the drum membrane, ossicles, etc., are designed to vibrate in an immobile head. The mere construction of the saccular receptor suggests that its stimulation involves an actual vibration or tremor of the head. We have no evidence that in any terrestrial vertebrate the saccular macula is commonly stimulated by tremor communicated to the body itself from the outside (exteroceptor). The production of voice by air-breathing vertebrates is liable to cause head tremor; therefore it is a fair inference that the saccular macula in these animals is a proprioceptor with the function of regulating the voice. We hear our own voices with two kinds of receptors, while we hear the voices of others with only one. (Note: In a number of phonograph records made by the author of the present work it has always been interesting to note that whereas the voice records very well it sounds not at all like the voice he daily hears.) Tait mentions this peculiarity of the phonographic record.

AMPHIBIA.

Urodela, an order of amphibians, with naked skin, usually two pairs of limbs and persistent tail (including salamanders).

ORDER 1.—*Perennibranchiata*, with persistent bushy, external gills and gill slits; maxilla usually lacking.

Sirenidae, hind limbs lacking. Siren, the mud-eel of Southern United States; jaws armed with horny sheaths.

Proteus, of Austrian caves; nearly blind.

Necturus (*menobranthus*), the mud-puppy of Central United States.

ORDER 2.—*Derotremata*, external gills lost, a spiracle on the side of the neck, leading to persisting gill-slits.

Menopoma (*cryptobranchus*), hell-bender, United States.

ORDER 3.—*Salamandrina*, gill-slits and external gills lost in the adult; vertebræ fully ossified. The genus *ambystoma*, remarkable for the length of time that their larvæ (*siredon*) retain their gills; some species, *A. tigrinum* and the axolotl of Mexico breeding in the *siredon* stage. Most of the lungless salamanders belong to this group.

Proteus anguinus, Laur., is a salamander-like amphibian, nearly blind and nearly colorless, with three fore and two hind toes and persistent external gills. It is found in Austrian caves.

Windischmann²⁸ found the aural capsule to be osseous. Within the capsule is a long cavity which narrows anteriorly. The floor is divided by a ridge into two parts: an anterior small one, containing an ampulla, and a larger posterior one, containing the "sacculus" and otoliths. The cavity has five walls showing four openings, namely, a fairly large oval one on the outer wall (*fenestra ovalis*), covered with a cartilaginous operculum; in the inner wall, three small openings for the passage of the three branches of the acusticus. There are three semicircular canals. Cochlea and *fenestra rotunda* are absent.

Kuhn²⁹ describes the capsule of the *proteus anguinus* as cartilaginous and osseous. At the end of the sacculus is the opening to the lagena. For the first time there appears a *pars initiales cochleæ*. This lies on the under surface of the floor of the utriculus and consists of a round cartilaginous vesicle. Its cavity contains a *membrana tectoria*.

Retzius found the membranous labyrinth of the *proteus* (Fig. 5-A) in many respects similar to that of fishes, particularly the ganoiden and teleostier. In common with the elasmobranchien it has the medially closed capsule.

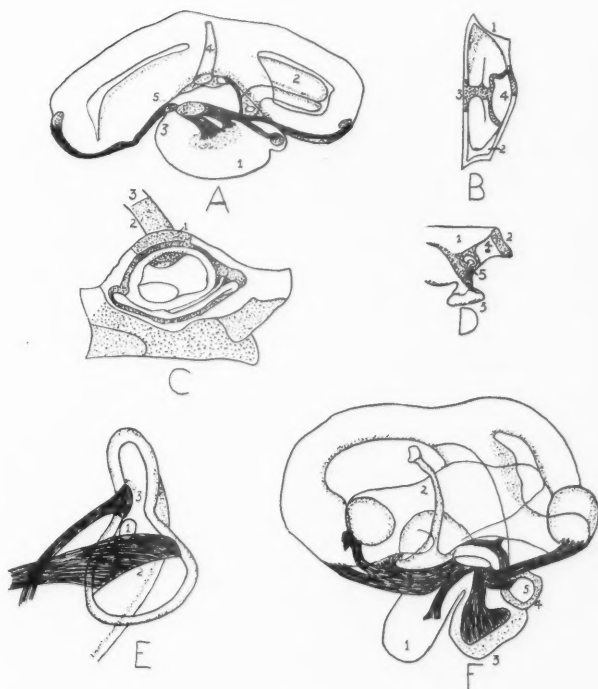


FIGURE 5.

A. *Proteus anguinus*, Laur. Membranous labyrinth. 1. Sacculus. 2. Utriculus. 3. Lagena. 4. Ductus endolymphaticus. 5. Ductus perilymphaticus. 6. Macula ac. neglecta.

B. *Proteus anguinus*, Laur. External wall of capsule from median side. 1. Occipitale laterale. 2. Prooticum. 3. Cartilage plate. 4. Bone corresponding to foot-plate of stapes.

C. *Menopoma alleghaniense*, Harl. Osseocartilaginous capsule from above. 1. Cartilage. 2. Osseous portion of columella. 3. Cartilaginous portion of columella.

D. *Menopoma alleghaniense*, Harl. Aural capsule—external view. 1. Parasphenoidale. 2. Occipitale laterale. 3. Squamosum. 4. Cartilage. 5. Columella with foot-plate in oval window.

E. *Menopoma alleghaniense*, Harl. 1. Opening into sacculus. 2. Papilla ac. lagenae with nerve. 3. Papilla ac. basilaris and nerve.

F. *Bufo vulgaris*, Laur and Hyla; arborea, Cuv. 1. Sacculus. 2. Utriculus. 3. Lagena cochleae. 4. Pars basilaris cochleae. 5. Papilla ac. basilaris.

—After Gustaf Retzius.

The lateral wall of the capsule of the proteus shows superiorly a division of the cartilage to surround a small bone (Fig. 5-B). This opening takes the place of the fenestra ovalis of higher amphibia, the operculum corresponding in a way to the "deckplatte" of the columella. On the median wall are four small openings: the apertura ductus perilymphatici, the apertura ductus endolymphatici, apertures for the ramus post. n. acusticæ and the ramus anterior n. acusticæ.

The membranous labyrinth is made up of utriculus with rudimentary sinus superior and a long sinus post., recessus utriculi, ampulla ant. with canalis ant., amp., extern. with canal, ampulla post. with canal, sacculus with ductus endolymph., lagena cochleæ and pars neglecta. The nerve endorgans are seven: three cristæ, maculæ for utriculus and sacculus, macula ac. neglecta and papilla ac. lagena coch.

In the canal joining the utriculus and sacculus is a recess containing the macula ac. neglecta. It has a long membrana tectoria of lamellar structure.

Siren lacertina, L.—This amphibian is called the mud-eel. The sirenia are an order of aquatic mammals of fish-like form, with the lower jaw as in ordinary mammals with molariform teeth for an herbivorous diet. The sirenia include manatees, dugongs et al.

In the membranous labyrinth one finds the utriculus with a short sinus superior and a long sinus posterior, recessus utriculi, three ampullæ and canals, sacculus with ductus endolymph., lagena coch., pars neglecta.

Menopoma alleghaniense, Harl.—The aural capsule (Fig. 5-C-D) is of cartilage and bone. On the lateral wall one finds inferiorly in the column of cartilage a large oval opening (foramen ovale), which is covered by the bony oval footplate of the columella. The footplate has a tendinous attachment to the edges of the oval window. This is the first mention of an annular ligament. The structure of the lagena is pictured in Fig. 5-E.

Siredon mexicanus, Bd.—These amphibia are really the larvæ of the amblystoma or blunt-nosed salamander. The larvæ reach a large size before the gills disappear, and they sometimes breed in the larval state. Formerly the siredon was considered a separate genus.

The vestibule shows on its inferior wall a fenestra ovalis. The posterior and larger part of the oval window is covered by a cartilaginous operculum; the remaining part, by a small cartilaginous patina or concave disc which is connected with a small bone. This bone articulates with the tympanicum.

Hasse³⁰ describes an evagination of the sacculus to which a small nerve passes and which shows on one wall a macula acustica. This evagination he calls the beginning of the cochlea. The columella is composed of three parts: pars externa, a bony pars media and a cartilaginous pars interna, the latter being attached to the oval window.

The labyrinth shows the beginning of the cochlea and the duct of the perilymphatic spaces. These spaces are very extensive; the greatest area being from the sacculus to the oval window. The perilymphatic duct passes through a round opening on the median wall of the capsule and into the cranial cavity, where it ends in the subdural space, allowing perilymph to mix with cerebrospinal fluid. The ductus endolymphaticus of one side joins that of the other, then expands into an enormous sac which extends over a good part of the brain.

Triton cristatus, Laur.—Scarpa³¹ found, in water salamanders, no outer canal, no drum membrane, no ossicles, no tympanum and no eustachian tube. He did find an oval window closed with a cartilage plate. Against the oval window lay the sacculus.

Retzius³² describes in the *triton cristatus* a true pars basilaris cochleæ.

Caccilia annulata, a limbless tropical amphibian of worm-like form, with no tail. Skull is well ossified. It burrows in the earth, preying upon small invertebrates.

Scarpa found drum membranes deeply situated under the general roof of the capsules. The cartilaginous process of the ossicle, which is shaped like the letter r, is firmly attached to the drum membrane. The bony process passes to the oval window and closes it. The tympanum is quite large. From it the eustachian tube passes to the pharynx. The eyes are extremely small, so small, in fact, that the observer, Scarpa states, is led to believe that the animal is almost without sight. He thinks this may

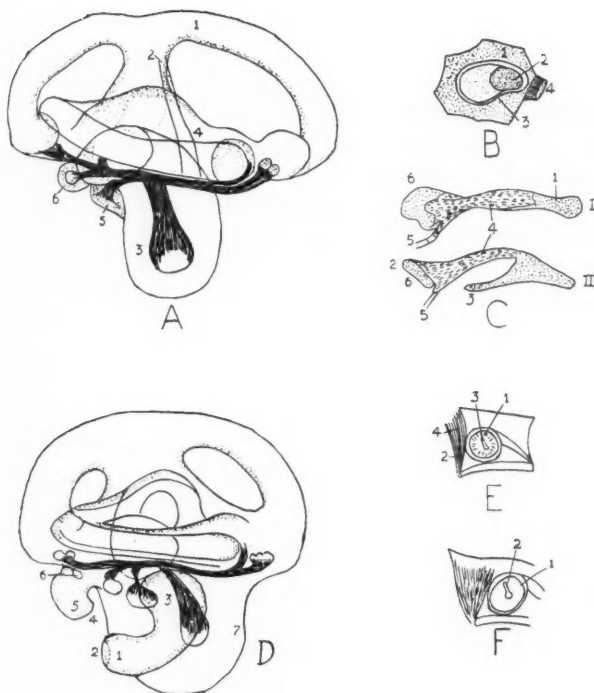


FIGURE 6.

A. *Rana esculenta*, L. 1. Canalis m. anterior. 2. Ductus endolymphaticus. 3. Sacculus. 4. Utriculus. 5. Lagena cochleae. 6. Pars basilaris cochleae.

B. *Rana esculenta*, L. 1. Osseo-cartilaginous wall of capsule. 2. Fenestra ovalis. 3. Fovea fenestrae ovalis. 4. Columella.

C. *Rana esculenta*, L. The columella. I. From superiorly. II. From posteriorly. 1. Outer end of cartilaginous part. 2. Upper inner short end, cartilaginous part. 3. Lower inner long end of cartilaginous part. 4. Osseous portion. 5. Small muscle. 6. Inner cartilaginous plate which covers the fovea fenestrae ovalis and the fenestra ovalis.

D. *Rana esculenta*, L. The membranous labyrinth of the frog from the lateral side, closed over by its periosteum. 1. Ductus fenestrae ovalis. 2. Oval opening leading from perilymphatic space to the ductus fenestrae ovalis. 3. Saccus fenestrae ovalis, ending blindly. 4. Ductus perilymphaticus. 5. Saccus perilymphaticus. 6. Canal leading to serous space of brain. 7. Sacculus.

E. *Rana esculenta*, L. Drum membrane, external view. 2. Attachment plate of columella. 3. Cartilaginous ring. 4. Musculus depressor maxillae.

F. *Rana esculenta*, L. Outer part of tympanum; drum membrane removed. 1. Opening through which the columella passes inward. 2. Attachment plate of columella.

—After Gustaf Retzius.

account for the ears being so much better developed than in other similar species. The capsule is of bone.

Anura.—Amphibia without tails in the adult state (frogs, toads, treetoads, etc.).

Bufo vulgaris, *Laur.*—The common toad has an aural capsule of cartilage and bone. The outer wall of the perilymphatic space is of thick connective tissue bearing pigmented cells. This wall forms the ductus fenestræ ovalis.

Hyla arborea, *Cuv.*—Treetoads have a membranous labyrinth (Fig. 5-F), comprising utricule with sinus superior and posterior, recessus utriculi, three ampullæ and canals, sacculus with ductus and saccus endolymphaticus, lagena cochleæ, pars basilaris cochleæ and pars neglecta.

Rana esculenta, *L.*—The edible frog.

Comparetti³³ was the first to describe in detail the ear of the frog. He mentioned the foramen ovale (Fig. 6-B), eustachian tube and membrana tympani with ring. To the membrana tympani is attached the outer broad cartilaginous key-formed end of the ossicle. Medially the ossicle covers the oval window with a partly cartilaginous, partly osseous plate.

Windischmann³⁴ could not find a cochlea or round window in batrachia. From the inner ear two passageways were found, ductus endolymphaticus and the following: in the inferior posterior part of the sacculus region is, in the vicinity of the oval window, an oval opening in the periosteal covering of the perilymphatic space. This opening leads into a passageway called the ductus fenestræ ovalis (Fig. 6-D). The passageway or canal passes outward and forward through the oval window between the inner cartilaginous portion of the columella and the fossa fenestræ ovalis (Fig. 6-B). It enlarges, passes upward and ends in the blind saccus fenestræ ovalis. The outer wall of this saccus contains pigment cells.

The ductus perilymphaticus passes above and posterior to the ductus fenestræ ovalis, posterior to the nervus acusticus; then posteriorly and superiorly from the lagena it passes out of a large round opening as a direct continuation of the periosteal wall of the perilymphatic space.

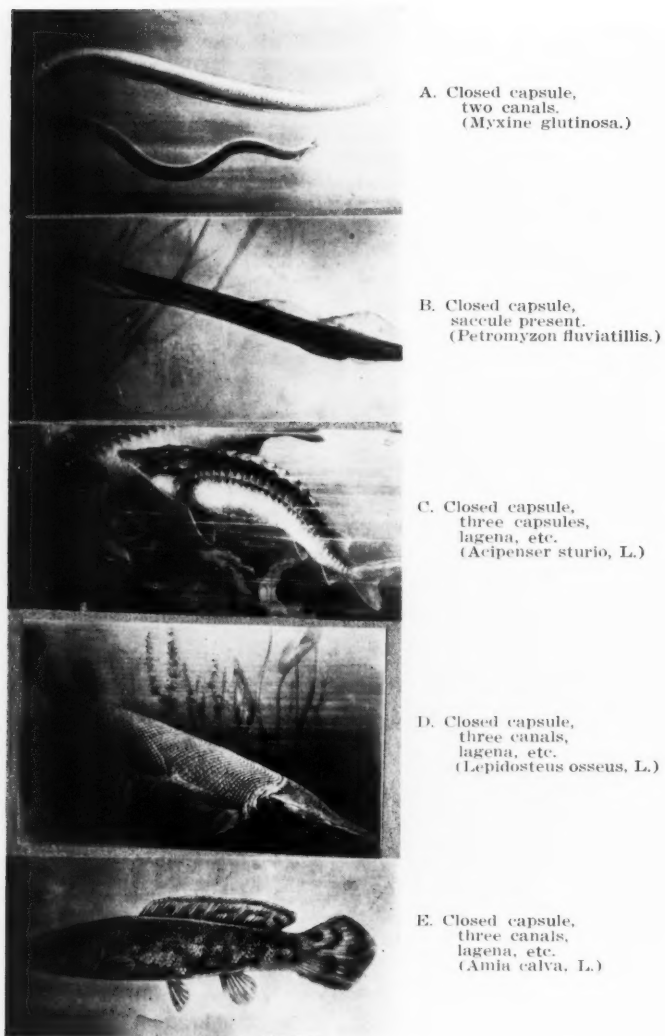


FIGURE 7.
(After Brehms Tierleben.)

Weber³⁵ showed that the frog has a fenestra rotunda, which is in the outlet of the canal through which the nervus vagus passes out of the cranial cavity. He also found that the round window is covered with a membrane.

Deiters³⁶ was the discoverer of the "cochlea" (Fig. 6-A) of the frog. In Fig. 6-C is pictured the columella; in Fig. 6-E, the drum membrane; in Fig. 6-F, the tympanum.

REPTILES.

Chelonia (tortoise).—Comparetti found in turtles two tympani. In the posterior one, the eustachian tube enters. Under the posterior tympanum is a long groove which broadens anteriorly to hold the base of the ossicle covering the oval window.

Scarpa described, in turtles, a small cartilage in the middle of the drum membrane. From the cartilage a bone extended and ended in a base covering the oval window. The tympanum was capacious and showed the opening of a short eustachian tube.

Windischmann found, in turtles, two windows: an anterior oval one with operculum, and a posterior larger, round window covered by the membrana tympani secundaria.

Retzius states that the ears of all the members of the turtle family are the same and that he agrees with the foregoing descriptions.

Ophidia (serpents).—In the viper, *Coluber Berus*, L., the adder, *Coluber natrix*, L., and certain large snakes from Asia, belonging to the adder family, no external ear, drum membrane, tympanum or eustachian tube were found by Geoffroy.³⁷ A long ossicle lay between the muscles moving the mandible and ended internally with a plate which covered the oval window and externally with a cartilage. No semicircular canals could be found.

Comparetti found in vipers a thin membrane covering the tympanum and two ossicles, a ligamentous and an osseous. The base of the osseous one covered the oval window and was attached by means of a strong membrane. Two semicircular canals were found.

Scarpa describes an ossicle covering the oval window of the viper. The pointed external end of the ossicle was attached to the supports of the jaws. He found no sign of a drum membrane, tympanum or eustachian tube. Three semicircular canals were found.

Siebold and Stannius²⁸ state that reptiles have two labyrinthine windows: fenestra rotunda for the cochlea and the fenestra ovalis. In some species the oval window is covered with a bony plate only, while in others an ossicle is attached. In most snakes the oval window is very broad.

Sauria (lizards, crocodiles).—Geoffroy examined the ears of different lizards and described the drum membrane, tympanum, ossicles, vestibule and three canals. The drum membrane is inserted into a large oval opening and is composed of an outer layer and an inner membrane continuous with the lining of the tympanum. Between the two is an ossicle corresponding to the malleus. This ossicle articulates superiorly with another which descends deeply in the tympanum to end as a plate in the oval window. The second ossicle, therefore, is a composite of the incus and the stapes. There is no external ear or canal, according to Comparetti. The tympanum is in wide communication with the mouth cavity, no tubular connection being present.

Windischmann found that in lizards the labyrinth lies partly in the petrosum and partly in the occipitale laterale. The small cochlear canal with its round window is in the occipitale laterale; the oval window lies in both. The drum membrane has three layers, as in man. Ossicula auditoria consist of three parts: operculum, columella and a cartilaginous part which joins the columella at right angles.

Von Clason²⁹ describes the osseous aural capsule of lizards as being formed by three bones: opistoticum, prooticum and epoticum—prooticum anteriorly, epoticum superiorly, opistoticum posteriorly. The cochlea rests in the opistoticum and epoticum. On the outer wall of the pyramid is the oval window. Anterior to the oval is the round window.

Retzius states that in the *Lacerta viridis*, Linn., and *Ocellata*, Daud, the perilymphatic space has for its outer boundary a thick fibrous periosteum studded with pigment cells. On one place, in the lateral periosteal wall between the cochlear and vestibular spaces, is a small, oval, ring-formed, unpigmented fibrous ridge. This ridge serves for the attachment of the footplate of the columella. No oval window is present.

Crocodylia.—According to Scarpa, the ears of the crocodile, lizard, frog, toad and turtle are alike except for the shape of the ossicle and its cartilaginous connection. In the crocodile the base of the ossicle is triangular. There are three semicircular canals. The lowest form of cochlea is present: a curved extension of the sacculus. It divides into two canals: one opens into the sacculus, the other ends with a small opening at the tympanum and is covered with a membrane.

The osseous labyrinth is thin but hard. The tympanum consists of two halves: an outer large space surrounded by bone and closed by the drum membrane; the inner half having the two openings, oval and round windows and several mastoid cells. The oval window is derived partly from petrosum and partly from occipitalis. The drum membrane is large and at its center is attached, with cartilage, the ossicle.

The operculum (footplate) articulates with the oval window in such a way as to be slightly movable. The bone surrounding the capsule contains large air cells.

Alligator mississippiensis, Gray.—The columella consists of an osseous inner portion and an outer triangular cartilaginous part. The inner end shows a thin oval bony plate which fits into the oval window. No muscle is attached to the columella. The drum membrane is of thin connective tissue covered on either side with epithelium.

BIRDS.

Comparetti found in certain birds of prey an outer membranous ear containing muscle and to an extent movable. The external canal varied in length and diameter as did also the drum membrane and ossicles. The round window was larger than the oval.

Scarpa⁴⁰ states that the inner ear of birds is divided into three parts: vestibule, canals and a sort of cochlea. To these lead two windows, round and oval. The round window is in relation to the cochlea and is closed with a membrane. The oval window is covered by the base of the ossicle. There are three canals with five openings into the utriculus.

Pohl⁴¹ found that certain birds show an almost triangular fenestra vestibuli; others the oval form. The entire labyrinth is surrounded by an air-containing diploe. The tympanum is broad and

divided by a bony ridge into an upper and lower part. The columella is in the upper part. The eustachian tubes either join in the pharynx or are very close together.

Breschet⁴² described the tympanum as having an outer and an inner part. On the superoposterior part of the inner surface of the inner part are the openings of the cellulae mastoideae. The tube is antero-inferior. There are two labyrinthine openings; one, the oval window, closed by the plate of the "stapes"; the other, the round window, closed by the membrana tympani secundarii. One ossicle with its processes represents all parts of the ossicular chain of mammalia, but only the "stapes" is osseous. The "malleus" has one muscle, a laxator tympani. There is just a suggestion of a stapedius muscle. The secondary tympanum is filled with fluid.

Hasse⁴³ found in the goose that the inner wall of the tympanum slants from posteriorly outward. A funnel-shaped space serves as receptor for the columella. Both oval and round windows are present. There are two types of tympanum in birds: (1) as represented in the chicken, suggesting the mammalian tympanum; (2) as seen in the goose, suggesting a transition from the reptilian and amphibian types. The higher type shows bony walls; the other has walls partly of soft tissue. In the higher type the oval window recess is deeper posterosuperiorly. The cochlear window is below the oval and is more oval than round.

MAMMALIA AND MAN.

Although the bony labyrinth was well understood by Eustachius, Faloppia, Vesalius, Casserius, Schelhammer, Duverney, Winslow, Vieussens, Valsalva, Cassebohm, Morgagni and Cotugno, it remained for Scarpa⁴⁴ to discover the membranous labyrinth and give us a better understanding of the cochlea. At the same time appeared Comparetti's description of the same parts.

In 1851, Corti⁴⁵ published his remarkable observations on the cochlea of mammals.

Breschet⁴⁶ examined the aural labyrinth of man, dog, cat, rabbit, pig, horse, deer, sheep and ox, and found in all a marked similarity, only slight differences being observed. In the dog, the

anterior canal is the largest; in the rabbit, the anterior canal is larger than the posterior, the posterior larger than the lateral; the cochlea has three turns. In the pig, the anterior canal is larger than the other two; the cochlea has four turns. The cochlea of the horse has two and one-half turns. In the rabbit the floor of the cisterna perilymphatica forms about 2 mm. of the lamina spiralis ossea and the membrana Reisnerri of the basal turn of the cochlea.

SUMMARY OF PHYLOGENETIC DATA.

In the lowest form of vertebrate life (Fig. 1-A, B, C) the *myxine glutinosa* (hag-fish), the aural capsules consist of bone and cartilage (the presence of bone is doubtful) and are completely closed, showing no evidence of oval or round window.

Next above comes the *petromyzon fluviatilis* (Fig. 1-D, E, F; Fig. 2-A, B), (river lamprey), with cartilaginous capsules and no openings corresponding to oval or round window. The membranous labyrinth, however, shows progression in that a sacculus (Fig. 1-E) has appeared.

Fishes have either cartilaginous aural capsules or a cartilage model replaced by a bony capsule in which islands of cartilage persist, as in man. When the cartilage model of man is hollowed out by osteogenic buds, bone is laid down in the spaces, but islands of cartilage persist and in these islands otosclerosis begins.

The sturgeon (*ganoidei*) (Fig. 2-C, D) shows a marked advance over the *petromyzon fluviatilis* in that it has three semi-circular canals, a lagena cochlea and three additional nerve endings, namely, one for the third canal, a macula neglecta and a nerve ending in the lagena. No oval or round window, however, has made its appearance.

The Breschet bone (Fig. 3-A), fixed between the sacculus and the capsule wall and looked upon as the homologue of the stapes or of all the ossicles, is a most interesting structure, first because it would seem to be a derivative of the Weber organ (chain of ossicles from swim-bladder to labyrinth), and therefore a part of the organ of equilibrium, and secondly because it is looked upon as the forerunner of the stapes, which we usually think of as having evolved to aid in the transformation of sound

waves for the cochlea: all this in the absence of oval or round window in the sturgeon.

When we reach the *teleostomi* (genuine bone-fish), we note that Weber found in the carp and catfish three ossicles articulating with the first three vertebrae. He named them stapes, incus and malleus, and found in the capsule an opening posteriorly which he considered the vestibular window. For the theory of regression the appearance of the vestibular window marks an important boundary, for the transmitted capsular character which results in otosclerosis must have originated below the level of this phylogenetic period. The membranous labyrinth is practically the same as in the *lepidosteus osseus* (Fig. 3-B).

In the herring (*clupea harengus* L.), Weber found the two labyrinths connected by a canal between the utriculi and passing under the brain. Breschet discovered a tubular connection passing above as well as under the brain. Because of the fact that in otosclerosis the involvement is almost always bilaterally identical, this connection between the labyrinths assumes importance in the relationship of morphology to otosclerosis; for it is easier to conceive of a regressive dystrophy involving the two sides of one organ symmetrically than of two entirely separated structures showing an identical involvement.

The description by Weber, Breschet and Haase of the first appearance, in the herring (*clupea harengus* L.) of a "tympanum" connected with the air bladder helps to substantiate the identification of the small, membrane-covered opening in the capsule as the forerunner of the oval window. It is interesting that this window does not appear as an independent structure but rather as a mere space left by the incomplete articulation of the prooticum, opisthoticum and occipitale basilare.

Retzius states that in the pike (*esox lucius*, L.) (Fig. 4-A) the occipitale basilare, occipitale superius, occipitale laterale, prooticum, epoticum and squamosum are joined by cartilage to form the labyrinthine capsule. These cartilage connections may represent the homologues of the cartilage partitions (see abstract of Leiri's article, elsewhere in this work) in which otosclerosis starts.

The short-snouted seahorse (*Hippocampus brevisrostris*, Leach), although far above the cyclostomata in that it has a lagena and

nerve ending for utriculus, sacculus, lagena and canals, shows a regression to the round-mouth period in its one vestibular cavity with rudimentary canals.

In the shark, external auditory meatuses first appear. An evagination of one angle of the triangular utriculus passes upward to end externally at the oval window. It is possible that this protrusion from the utriculus (accessory endolymphatic duct?) to the oval window is the origin of the vestigial structure named by Siebenmann, *fissula ante fenestram*. The *fissula* in the human, a passageway from the *cisterna perilymphatica* to the tympanum, situated anterior to the oval window, is present in all human embryos and in a certain number of adult temporal bones. In the *fissula* are protruded fibers from the endost of the *cisterna*. It is quite conceivable that in the course of evolution the connection with the utriculus could have been obliterated. This vestigial structure has no known function in the human but is of importance because, in it, otosclerosis often begins. In the thorn-shark, a one-piece aural capsule of hyalin cartilage, comparable to the cartilage model of the human capsule, makes its appearance. In lower phylogenetic strata we have learned that the aural capsule is made up of several cranial parts in articulation.

Although not of direct interest to the present research, the communication of the posterior canal with the sacculus in the *squatina angeles*, or angel fish, and of the anterior and exterior ampullæ with the sacculus of the mud-fish, will be food for thought to those who believe the sacculus is an organ of hearing rather than of equilibrium.

The ray (*Batoidei*) presents a complete aural capsule of cartilage, entirely independent of the cranium, and an oval window (Fig. 4-F), covered with a membrane. The ray, according to Scarpa, is the first vertebrate to show a drum membrane. Also in this class is found a canal from the utriculus passing not only to the oval window, as in the shark, but actually through it. Here we have a more striking resemblance to the *fissula ante fenestram*, which passes from the *cisterna* to the tympanum.

In the *lepidosiren*, or lung-fish, we note on one hand the advanced amphibious trait of being able to obtain oxygen in the

water or, in dry season, entirely from air, by breathing exclusively with its lungs; on the other hand, a most primitive aural apparatus showing no external canal, tympanum, eustachian tube, lagena or windows.

In the first order of amphibia is the *proteus anguinus*, which arouses our interest, because it is the first vertebrate to show a "stapedial footplate" in the form of an osseous operculum (Fig. 5-B), covering the oval window. No crura or other evidence of a true stapes have been described. The existence of a footplate without crura makes us recall the fact that in man the footplate is originally a separate structure, developing from the capsule and only later blending with the crura to form a stapes of one piece. The *proteus* is of further interest because it shows for the first time a pars initiales cochleæ with a membrana tectoria. A round window has not yet appeared.

The *menopoma alleghanniense* possesses a footplate attached to the oval window by means of an annular ligament (Fig. 5-D); connected to the footplate is the homologue of the ossicles, a columella. We have then, here, a morphology closely related to that of man.

In the limbless amphibian, *caecilia annulata*, the aural organ is more highly developed than in any of the previously discussed vertebrates. The capsule is of bone: drum membrane, ossicle, oval window, tympanum, eustachian tube, are all present.

In the advanced development of the frog's ear, it is interesting to note that Comparetti found the inner end of the ossicle (Fig. 6-C), which covers the oval window, composed as in the human of cartilage and bone.

The ossicle shows three parts: an outer cartilaginous portion attached to the drum membrane, an inner cartilaginous and osseous portion articulated to the oval window; a middle portion, composed of bone showing, as do the malleus and stapes of the human, a small muscle attachment. Another structure in the frog's ear, the ductus fenestræ ovalis, engages our attention. It emerges from the perilymphatic space near the sacculus, passes outward between the columella, footplate and the oval window fossa, to end blindly, superiorly and anteriorly in a broad expansion, the saccus fenestræ ovalis. It may be that the fissula ante

fenestram is the vestige of this duct. Inasmuch as both structures start in the perilymphatic space and pass through to the tympanum they probably are more closely related than the previously mentioned protrusions of the utriculus are to the fissula. It would seem that in amphibia and mammalia an accessory endolymphatic duct is no longer necessary.

In the frog there appears for the first time a true round window covered with a membrane. The turtle also has an oval window covered by an operculum (footplate) and a round window covered by a membrana tympani secundaria.

In the snakes examined by Geoffroy, an ossicle was found between the muscles moving the mandible. Externally the ossicle ends in cartilage, internally with a plate covering the oval window. In the chapter on ontogenesis of the human ear will be given a description of a related structure, Meckel's cartilage (cartilage core of the mandibular portion of the first branchial arch) which forms the malleus and incus. The cartilage later separates, through resorption, from the ossicles and finally disappears from the mandible.

In certain lizards two ossicles are present. One lies between the two layers of the drum membrane and corresponds to the malleus; the other, articulating with the first, ends with a plate which covers the oval window. Although in man the stapes and incus are derived from two different arches, in these lizards the one ossicle is a composite of both stapes and incus.

In the lizards, *lacerta viridis* and *ocellata*, Retzius found no oval window but a ring-formed fibrous ridge in the periosteum which served for the attachment of the footplate of the columella. This regressive trait is of interest in connection with the regression in otosclerosis. One wonders why the footplate attachment with no window through which sound could be transmitted to the labyrinth unless it be that sound waves are here transmitted through the bone by movement of the footplate.

In birds, the oval window is above the round window as in man. There are two types of tympanum; one is similar to man's and is represented by the chicken's ear; the other is a transitional form, having certain characteristics of the reptilian and amphibian ears, and is represented by the goose's ear. Unlike the human

labyrinthine capsule, the bird's membranous labyrinth is surrounded by a comparatively thin, compact portion, the remainder being of diploe.

(TO BE CONTINUED.)

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Neoplasms of the Nose, Throat and Ear.

CARCINOMA OF THE HYPOPHARYNX.

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Carcinoma of the hypopharynx includes all malignant epithelial neoplasms originating in the area lying between two planes, one passing horizontally through the inferior portion of the base of the tongue and the other passing parallel to the first, through the mouth of the esophagus. Designating them anatomically, these various sites may be the posterior inferior portion of the tongue, the epiglottis, the valliculæ, the pyriform sinuses, the lateral and also the posterior pharyngeal wall, the postericoid and interarytenoid region, and last the aryteno-, pharyngo- and glosso-epiglottic folds.

It has been considered convenient and logical, from a clinical point of view, to divide hypopharyngeal growths into those located in the upper hypopharynx, the so-called epilaryngeal group; those in the midportion of the hypopharynx, the so-called postericoid growths, and finally those of the lateral portion of the hypopharynx, the so-called pyriform sinus growths. From a histologic point of view, these neoplasms most frequently are of a squamous cell type and less frequently one may observe the so-called transitional cell carcinoma and the lympho-epithelioma. Dissemination of the growth to the regional lymph glands is a constant finding. Most frequently the carotid and superclavicular glands are involved and much more rarely one may find the mastoid gland similarly affected.

An important clinical consideration is the fact that digital examination may not disclose any palpable glandular involvement and yet microscopic evidence of the disease in the lymphatic glands in the neck may be present. Another important clinical fact to

bear in mind is that frequently large, massive glandular metastasis in the neck may be the first manifestation of a very small hypopharyngeal growth that of itself gives rise to no subjective symptoms and may even be difficult to detect by endoscopic examination. In such instances one will most often find the histologic type of the disease to be either the transitional cell carcinoma or the lympho-epithelioma.

A third important clinical observation must also be stressed. Not all palpable enlarged cervical glands are necessarily malignant. Experience has shown that sometimes as much as from 10 to 20 per cent of these palpable glands may be infectious and not carcinomatous in nature. The clinical course of the disease varies markedly with the site of origin of the malignant growth as well as its histologic type. As these factors have a very important bearing on the treatment, a few brief statements will be made relative to the various sites of origin.

1. Aryepiglottic malignancy is well known and described as the so-called extrinsic carcinoma of the larynx. In view of the fact that hoarseness and even dyspnea can be present with moderate sized growths, patients once afflicted come to seek medical aid early before extensive growth of the neoplasm and dissemination into the neck occur. These factors are important in so far as they permit observation of the patient while in an early operable condition.

2. Pyriform sinus growths, on the contrary, are but rarely seen during their early small localized stages of development. The location of these growths in the pyriform recess causes no difficulty that is detected on the part of the patient until they have reached sufficient size to encroach upon the pharyngeal lumen and thus cause some local discomfort that may increase with the growth of the tumor until actual dysphagia is produced. In these cases marked cervical glandular enlargement may be present before dysphagia or even discomfort may be complained of, and it is most frequently found that the neoplasm is of the so-called transitional or lympho-epithelial variety.

3. Malignancy of the lateral pharyngeal wall is one of the most important of the hypopharyngeal growths. It starts at about the

level of the arytenoid, and by increasing in size extends until it involves the entire lateral wall of the larynx and the arytenoid. Not infrequently the growth may be of a fungating nature, which will project and hang over the lateral portion of the larynx and may thus be mistaken for either a pyriform sinus malignancy or aryepiglottic growth. The clinical distinction between the sites of the growth are decidedly important in determining the details of the treatment.

These growths histologically are most frequently adult, more or less fully differentiated, squamous cell carcinomata and are characterized clinically by slow growth and progression. Their anatomic situation is also exceptionally favorable for excision by means of a lateral pharyngotomy, unless the growth by extension has finally invaded the larynx, when operation is contraindicated. Extension to the larynx is evidenced clinically by hoarseness and fixation of the vocal cords. As long as these two signs and symptoms are absent in a lateral pharyngeal wall carcinoma, the lesion should be viewed as operable.

4. Posterior pharyngeal wall malignancy is relatively rarely seen clinically. These growths are peculiar in that a large percentage of them are found in women. They are also favorably located for surgical resection and unless very extensive infiltration has occurred they should be looked upon as operable.

5. Postericoid malignancy is unusual in that it is a neoplasm most frequently found in women and during relatively early periods in life. The location of a carcinoma in this area is essentially one that is accompanied by symptoms. Even in the early stages of development of a mass in this area some degree of dysphagia is always found, and this difficulty in swallowing, of course, increases with the extent of the growth.

SIGNS AND SYMPTOMS.

The symptoms associated with hypopharyngeal carcinomata during the early stages are vague, as a rule. Later in the development of the lesion discomfort in the throat may be complained of and this may gradually increase in severity until actual dysphagia is present. Examination with the indirect mirror may

frequently show nothing, although not unusually one may observe a collection of mucus in the pyriform sinus that does not disappear on attempts at swallowing. At other times a small ulceration or even a neoplasm may be seen when the patient is made to phonate strongly. Another not unusual sign that is present is some edema of the arytenoid. The diagnosis is mainly made by the use of the endoscopic tube. Specular examination is depended upon not only to make the diagnosis but also to obtain a specimen for histologic study of the neoplasm causing the pharyngeal discomfort or dysphagia.

The X-ray diagnosis of neoplasms of the hypopharynx has been neglected in the past. Recently, however, Coutard and Baclesse¹ have shown that lesions located in this area may well be found by X-ray examination. In their studies they have found that the normally transparent areas and clear shadows of the hypopharynx as pictured in the usual X-ray film may be seen distorted or obscured by shadows due to infiltrating and fungating neoplasms. By repeated X-ray examination they have been able to follow regressions of growths located in this area when subjected to irradiation. The use of the fluoroscope with the barium meal may at times give exceedingly valuable information, especially if the fluoroscopy is performed with the patient in the supine posture, as in this position the barium is enabled to pass the area under observation very much more slowly than in the upright one.

The prognosis of carcinomata of the hypopharynx, while unfavorable, has undergone a distinct material change from the hopeless attitude of the past. This in the main has been due to the efforts of Trotter² and other workers, from the surgical point of view, and Coutard's³ work with roentgen ray therapy. Adult squamous carcinoma that is still confined to the pharyngeal wall with no glandular involvement offers a relatively good prognosis. The presence of small firm and nonadherent glands do not in themselves create a hopeless prognosis from a surgical point of view unless, however, the lesion is a postericoid carcinoma of the lympho-epithelial or transitional cell variety. The presence of marked cervical adenopathy in which the glands are decidedly enlarged and matted together offers a much worse prognosis.

As far as the clinical location of the disease is concerned, those located in the aryepiglottic fold or the lateral pharyngeal wall offer the best prognosis from the surgical standpoint.

TREATMENT.

In consideration of the treatment of carcinoma of the hypopharynx one should ever bear in mind that these conditions, permitted to progress untreated, are attended by a mortality of 100 per cent. While the results of surgery and irradiation therapy are not at all brilliant, at least they offer some ray of hope and may act as a guide in devising some means of improving our results in the future. Trotter² has at least seven cases of carcinoma of the hypopharynx alive and well and free from recurrence for a period of five to twenty years after surgical intervention, and Coutard³ had nine cases alive and free from recurrence from five to nine years after treatment by X-ray therapy.

The consideration of what method of therapy to employ, either surgery or irradiation, is still open to discussion and at the present time is somewhat difficult to answer. There is no difficulty encountered in those cases that are frankly and obviously inoperable. These must of necessity be treated by irradiation, with roentgen ray therapy or radium. A rapidly growing and early disseminating transitional cell and lympho-epithelial neoplasm in all probability should also be preferably treated by irradiation.

Treatment by surgical excision consists of the removal of the growth by procedure of transthyroid pharyngotomy, an operation that permits of surgical access to the entire laryngopharynx. A thorough resection of the gland-bearing area may be performed previously or, if the glandular involvement is not extensive and the patient's general condition permits, both the glandular resection as well as the pharyngotomy may be performed at the same time. Before any surgery is contemplated adequate consideration must be given to the patient's general systemic condition.

The English writers devote a great deal of attention to dental sepsis in these unfortunate individuals, and they frequently insist that they be rendered edentulous before any major surgery is performed. A preliminary tracheotomy is necessary, and it is to

be pointed out at this time that this step should be performed as low down on the neck as possible, and the incision should be made in the horizontal manner rather than in the classical vertical one. These measures are necessary in order that the formation of a skin flap, so essential to the success of the operation, may not be interfered with. The skin incision, which is such an important factor in the formation of the various flaps, forms so that it faces one side of the neck and its distal portion will extend beyond the midline of the neck to the opposite sternomastoid. The incision should start below the angle of the jaw and just above the clavicle. The flap that is reflected includes not only the platysma but also the subcutaneous tissue as well, and at the base a portion of the sternomastoid muscle is included also. The deep fascia and the gland-bearing tissues of the neck are then resected, exposing the carotid sheath and the perilaryngeal muscles. If these glands are extensively involved the internal jugular vein and at times even the carotid artery itself may have to be resected. If the vessels are left undisturbed they should be protected from subsequent exposure to infection by suturing the remaining portion of the sternomastoid muscle to the prevertebral musculature. One may also utilize the pretracheal muscles and the lobe of the thyroid, which should be freed and then placed across the vessels and sutured to the remains of the sternomastoid. These procedures afford some degree of protection after the esophagus has been divided. The thyroid cartilage and at times the hyoid bone is then laid bare on the affected side, by resecting the overlying musculature and the greater portion of the cartilage and, if required, part of the hyoid bone is removed, thus exposing the pharyngeal aponeurosis. The pharynx is then palpated and note is made of the limits of the growth, and a decision is arrived at whether or not the growth is operable. In reaching a decision as to the operability of the mass, it is well to bear in mind that if the neoplasm has extended far into the oropharynx or below the cervical esophagus or has progressed beyond the signet portion of the cricoid cartilage and invaded its lateral aspects, it should no longer be considered operable. If at this time it is determined that the growth cannot be well resected through this means and that further surgical intervention is not feasible, it is probably

best to either resort to the use of deep X-ray therapy or irradiation therapy with radium.

If radium is decided upon it is well at this time to thoroughly irradiate the entire growth and the surrounding area by the introduction of one and two milligram needles of radium screened by 0.5 millimeters of platinum throughout the entire area. The flap that has been previously made should be protected by the use of a sheet of lead foil. The radium is permitted to remain in situ for a period of five to seven days and is then removed, the lead foil being taken away at the same time.

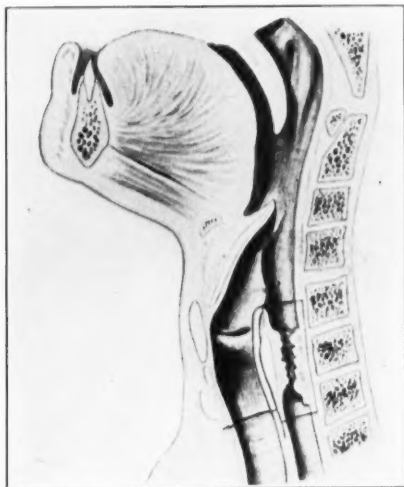


Fig. 1. Seifert's operation showing incisions for the removal of circular growth.

If it is determined that the growth is operable the procedure is continued by packing off the lower portion of the neck. The pharynx is then incised well up beyond the upper limits of the growth and the neoplasm is resected, the surgeon staying well within the healthy tissue. When a portion of the pharynx and its contained growth is removed, there remains a large defect extending from the lower border of the pharynx itself to the upper border of the esophagus below. This defect is repaired by utiliz-

ing the skin flap mentioned before. The upper edge of the flap is sutured to the lower pharyngeal border, and the lower edge of the flap is sutured to the esophageal border of the defect. The free end of the flap is then curled so as to form a trough extending from the pharynx itself to the esophagus below and is sutured in place. (Fig. 1.) The esophageal feeding tube is introduced and dressing applied and the procedure terminated. The trough that is formed to take the place of the pharynx gradually becomes converted into a deep gutter. Later the skin forming the anterior

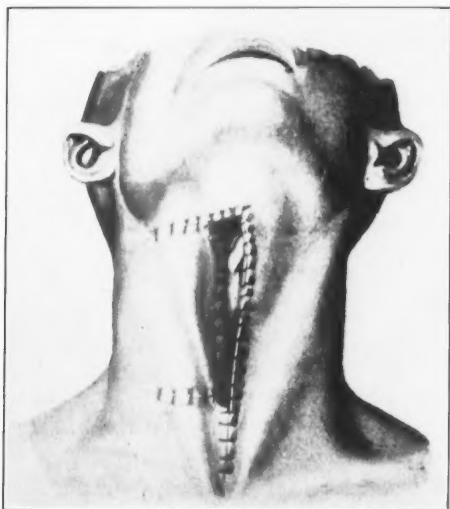


Fig. 2. Formation of trough after resection of hypopharynx.

and posterior borders of the gutter may be incised, utilizing a little local anesthesia, and the inlaid skin flap sutured in the form of a tube over the feeding tube. The opposing skin edges that remain may then be sutured over the defect. By this means the resected pharynx has been replaced by a dermal tube. (Fig. 2.) When healing has occurred over a period of time, the feeding tube is removed and the patient can swallow via the skin-lined pharynx.

IRRADIATION THERAPY IN MALIGNANT DISEASE OF THE
HYPOPHARYNX.

Mention has been made of the use of radium element, well screened and in relatively small amounts over relatively long periods of time, in the preceding portions of this paper. Radium may also be utilized for the condition that is under discussion by means of a large amount at a relatively long distance from the skin, the so-called telioradium therapy, or the four-gram radium "bomb." This method is being employed in a large number of clinical centers throughout the world. However, in highly adult types of malignancies the amount of irradiation that can be brought to bear upon these deeply situated neoplasms by means of the radium bomb itself may be insufficient, and so in addition we advocate the use of interstitial irradiation, either with the actual element, in the form of needles distributed throughout the lesion or the use of gold or preferably platinum seeds containing radon gas. Peroral introduction of radium, either of the element itself or radon seeds, is rarely, if ever, curative and we only resort to its use in hopeless cases. We prefer to expose the growth either by the use of the Haslinger directoscope or one of the larger Haslinger hypopharyngeal tubes. The seeds containing the radon are then evenly distributed throughout the lesion. Not rarely the entire lesion must be reseeded after an interval of three to five weeks. We have discontinued the direct application of contact radium to lesions within the oral, pharyngo- and laryngopharynx, mainly because of the technical difficulties in keeping the radium in proper contact with the lesion and also for the reason that it does not permit of adequate doses of the gamma ray.

The most popular and by far the most effective method of utilizing radium therapy in malignant disease of the hypopharynx consists of its actual introduction into the neoplasm and the surrounding tissues. One must, however, utilize the so-called surgery of approach, and this consists of nothing more or less than of the lateral transthyroid pharyngotomy described above, except that the pharynx itself is not opened. After exposure of the pharynx and the tumor-bearing area the lesion and its surrounding tissue

is well needled with the radium element in the form of one and two centimeter length needles. These are well screened by at least 0.5 millimeters of platinum and permitted to remain in situ for over a period of five to seven days, when they are removed. By far the most important technical advance in the use of irradiation therapy in hypopharyngeal malignancy consists of a new X-ray therapy technic as evolved by Coutard,³ the so-called fractional dosage method. By this new means of therapy Coutard has been enabled to obtain an exceptionally high percentage of cures that in former years have been 100 per cent fatal. Coutard has based the philosophy of the therapy on the observation that the radio sensitivity of the cancer cells that are of epithelial origin are usually of the same degree of radio sensitivity as the germinal cells of the epithelium itself. Reasoning from this fact, if one can effect the complete destruction of the germinal cells of the epidermis of the irradiated surface without actually seriously injuring the true skin or dermis, any epithelial malignancy situated in the same plane and in the center of the irradiated surface would of necessity also be destroyed. Clinical experience at the Curé Institute has shown the truth of the principle stated above. Coutard then believes in giving sufficient doses of irradiation to cause destruction of the germinal cells of the cancer-bearing mucosa.

The resulting radio inflammation is called a radio-epidermitis when the lesion affects the skin, and radio-epithelitis when the lesion affects the mucous membrane. The dose that is required to produce the radio-epithelitis of the mucous membrane or a cutaneous radio-epidermitis is known as an epidermicidal dose. Radio-epithelitis of the mucous membrane presents itself clinically by the formation of whitish plaques of false membrane in the mucosa appearing about two weeks after the beginning of irradiation. The radio-epithelitis lasts for a period of about two weeks, and thus within a period of a month after the beginning of the irradiation the mucous membrane undergoes a cycle of inflammation, exudation and healing. At about the end of the fourth week the radio-epidermitis of the skin appears (Fig. 3), and is indicated clinically by redness, desquamation and then denudation of the skin. The repair occurs by the proliferation

of peripheral epithelial cells situated at the border of the defect, and the continuing development of the repair gives a polycyclic appearance to the edge of the lesion. It takes another period of about two weeks for the repair of the skin to occur. Thus one can see that within two weeks after the beginning of treatment the first signs of radio-epithelitis appear within the pharynx.

The subsequent formation of the exudate, its desquamation and healing occupy the third and fourth week following the irradiation. At the end of that period the radio-dermatitis and the

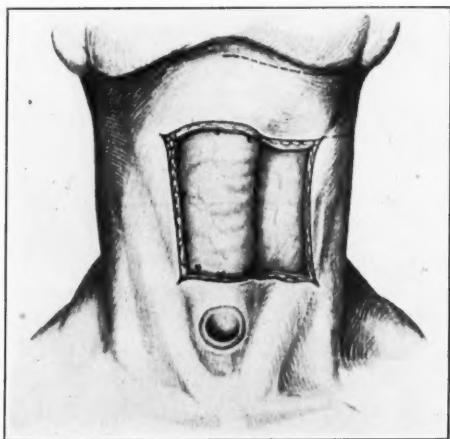


Fig. 3. Pharyngolaryngectomy: The skin flap is turned inwards and sutured in place, thus completing the formation of the reconstructed esophagus.

lesions of the skin appear, and its cycle of inflammation, denudation and repair occupies the fifth and sixth weeks after irradiation. Coutard has found that the duration of the evolution of the radio lesions described above constitutes an exact control that the irradiation therapy has been correctly applied. When the periods of evolution of the two distinct types of lesions overlapped it was a definite biologic indication that the irradiation has been incorrect, because either the doses have been too large or because the time of the treatment has been too short. On the contrary,

when the periods of evolution of the two radio lesions that were described were definitely separated the irradiation was also incorrect. In this case the doses were either too small or the periods of treatment were too long. Coutard has found that one dose of 4500 to 5000 R. distributed over a period of ten days in two daily sittings of one hour each for a total of twenty hours, over the same lateral surface of the neck, causes the appearance of the cutaneous and mucous membrane lesions noted above.

Physical constants under which the dose is administered is as follows: A focal distance of fifty centimeters; field being fifty square centimeters; with four milliamperes; a hundred and seventy-five kilovolts; with a filter of two millimeters of zinc; using a constant potential generator. In most cases after the administration of the above mentioned dose, the lesion will disappear after a period of about three or four weeks.

It is important to keep the patient under constant observation during this period of treatment because the patient very rapidly undergoes extreme loss of strength and weight. Fluids must be forced to the extreme and resort to the use of hypodermoclysis, or even intravenous infusion at times, is necessary. The use of the high carbohydrate diets may also be of some assistance. Progressive disappearance of the lesion may be carefully watched by endoscopy, and especially by the flat X-ray plate, as mentioned in the earlier portions of this article.

After the lesion has entirely disappeared repeated X-rays of the neck and pharynx at an appropriate interval are to be advocated, as by them at times a very early recurrence may be noted. While it is true that a large number of patients will have recurrence and not a few suffer severe reaction from this rather heroic treatment, the fact remains that a fair percentage of cures is possible.

That this method is the most promising one at our command today can be attested by the large number of favorable reports in its use that were presented before the German Otolaryngological Society held at Bad Elms in May, 1932. A review of these reports⁴ shows that Jung and Martenstein obtained healing in about 18 per cent of their cases subjected to irradiation therapy. Huenermann concluded that the Coutard method of therapy was a dis-

tinct advance in the method of treatment in spite of the disadvantages of expense and the time required. Thielmann was enabled to hold many of his patients symptom free for a period of one or two years, even permitting some to pursue their routine work. Richter, reporting upon the experience at the Erlanger Clinic, concluded that operable cases should be subjected to surgery first and then to follow with irradiation therapy. He apparently was well impressed with the possibilities of the Coutard method, but since they were employed for only a period of two years did not include them in his report. Krause obtained better immediate results with the Coutard method, but admitted that they were too recent to permit of judgment of the end results. Hesse believed that it was too early to properly evaluate the influence of the Coutard technic and recommended surgical removal of accessible growths as more certain. Mittermaier concluded that irradiation therapy is at least temporarily successful in the more radio-sensitive growths, but advocated surgical removal by diathermy before raying the growth. Schinz, in the discussion of the papers, concluded that the temporary results were better by far by the Coutard method. Holfelder was unable to see any advantages of the method over other accepted methods of irradiation. Halberstadter called attention to the late sequelæ, appearing months or even years after irradiation therapy, in the nature of telangiectasia, skin atrophy, ulceration and even necrosis. Hedfeld did not believe that the Coutard method would be successful in deep-lying neoplasms, because the passage of the rays through the tissues surrounding the tumor would damage the normal cells and impair their resistance to the neoplasm. He advocated irradiation to the pathologic area only. Marshik was convinced that the best results were obtained by following surgery by irradiation except in hypopharyngeal growths, which should be treated by irradiation solely. Vogel disagreed with Richter that vocal cord malignancy could not be irradiated. He believes that irradiation should be tried first and surgery used later if necessary. Brock did not agree that unsuccessful irradiation of a tumor could be followed by a successful second irradiation and quoted a case in which the primary irradiation was unsuccessful while a second series of treatments caused the growth to disappear. Seifert has been using

the Coutard method for a period of two years, mostly on inoperable cases with glandular metastasis. Some of the cases that were autopsied showed that the primary lesion had healed. He advised irradiation of the glands followed by operative removal after they had shrunk in size. Wessely believes that while the Coutard method has shown improved results they were only palliative, and advised that only five-year cures should be included under statistics. Mayer believed that the Coutard method was the best method of treatment of hypopharyngeal carcinomata today. Hofer believed that as surgery was unsatisfactory for tonsil and hypopharyngeal malignancy irradiation was preferred. In cancer of the larynx irradiation was insufficient. Hirsch had a satisfactory experience with the Coutard method over a period of two years. Albanus believes that the radio-epithelitis is a poor guide to dosage. In closing, Holthusen stated that the method was not as important as the dosage, and while permanent results cannot be claimed, the results so far are far superior to previous ones. Hegener closed by stating that if irradiation gives these unfortunates as much as two years of comfortable life it was worth while.

Thus it can be seen that the exact value of Coutard's method of X-ray therapy cannot as yet be determined, as time is the essential element. In the next ten to fifteen years statistics will definitely determine any value that it may have.

In conclusion, it should be stressed that hypopharyngeal malignancy is not altogether a hopeless condition. If the lesion is seen early, when it is small and localized, especially if it is of the adult, fully differentiated type, some hope is present that the patient can be subjected to a successful lateral transthyroid pharyngotomy. If the lesion is diffuse and has already spread to the lymph glands, and especially if the histologic type be that of a highly radio-sensitive one, the use of deep X-ray therapy, after the method of Coutard, or the use of irradiation, by either the four-gram pack or the regular interstitial application after surgical exposure, opens up a much more hopeful outlook for these poor unfortunates.

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Geo. Kennedy

GEORGE FETTEROLF, M. D., Sc. D.

1869—1932.

The passing of George Fetterolf on the twenty-ninth of last December closed the life service of a most brilliant and scholarly otolaryngologist. He had attained distinction both as an anatomist and as an otolaryngologist and had honored both of these departments of medicine.

Dr. Fetterolf was born at Collegeville, Montgomery County, Pennsylvania, the son of Adam H. Fetterolf, A. M., Ph. D., LL. D., who subsequently became President of Girard College in Philadelphia. As a child his sole ambition was to practice medicine and his brother recalls that he obtained from the family doctor and carried about bottles from which he dosed his playmates with salt and pepper powders.

Following an early preparation at Episcopal Academy, Philadelphia, he entered the University of Pennsylvania at the age of 14, graduated with the distinguished Class of 1887,—then followed a year of postgraduate work in the Biological Department. He received his medical degree from the University in 1891, Honorary Degree of Doctor of Sciences from Ursinus College in 1911, and held the title of Major in the Medical Corps of the United States Army in 1918-1919.

Three quotations crystallize the high opinion in which this able man was held. The first is an excerpt from the resolution adopted by the University Faculty of Medicine on January 3rd of this year—"Twenty-three years in the Department of Anatomy, in which for nine years he served as Assistant Professor. Thus he laid that foundation in fundamental science upon which he developed his knowledge and skill in his chosen specialty. He was elected Professor of Otolaryngology in 1924, thereby combining under his leadership the two formerly separate chairs of Otology and Laryngology."

The second follows from a Minute adopted by the Board of Trustees of the University on January 12th: "Dr. Fetterolf contributed many important studies bearing on the Anatomical relations as applied to Clinical Medicine in its various fields."

The final quotation is from an article on "Famous Men Who Have Held the Chair of Anatomy," by Dr. deSchweinitz in the *Pennsylvania Gazette*: "It is interesting to record that five of these eight professors of Anatomy were surgeons noted for their skill, an indication of how greatly operative technic, dexterity and judgment depend upon intimate acquaintance with regional anatomy; and the same is true of many of those who during the last sixty-five years served the Anatomical Department as demonstrators and assistant demonstrators. It is only necessary to mention a few of these men whose surgical and operative ability was so largely the outcome of their anatomical studies and dissecting room experiences: William Hunt, D. Hayes Agnew, Charles Hunter, Charles B. Nancrede, H. Lenox Hodge, John B. Deaver, Richard Harte and George Fetterolf."

Few men have more wholly devoted their lives to teaching service. Dr. Fetterolf commenced as demonstrator of Histology and Embryology in the Biological Department of the University in 1887. Then followed the positions of Prosector to the Professor of Applied Anatomy, Assistant Demonstrator of Anatomy, Demonstrator of Chemistry, Acting Demonstrator, Demonstrator, Instructor and finally Assistant Professor of Anatomy. His affection for and close association with Dr. George A. Piersol, who held the chair of Anatomy from 1898 to 1921, is a striking memorial to that service.

Equally arduous were his positions in his chosen specialty: Clinical Assistant in the Ear Department of the Polyclinic Hospital, 1893-1896; Assistant Laryngologist to the Methodist Episcopal Hospital, 1907-1910; Laryngologist to Henry Phipps Institute, 1905; Laryngologist to White Haven Sanatorium, 1906; Assistant Laryngologist to the University Hospital, Consulting Laryngologist to Eagleville Sanatorium, Eastern Pennsylvania Institute for the Feeble Minded and Epileptic, Kensington Hospital and Dispensary for Tuberculosis, Phoenixville Hospital and the Shriners' Hospital for Crippled Children. This chapter of generous and public spirited service proves how well he deserved his appointment to the Professorship of Otolaryngology at the University of Pennsylvania, which he served with distinction from 1924 until the day of his death.

We find yet another added service—membership on the Editorial Boards of the *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* and the Archives of Otolaryngology, the contribution of important chapters in Piersol's *Anatomy*, Hare's *Modern Medicine*, and Jackson and Coates' "The Nose, Throat and Ear and Their Diseases," and the preparation of the long series of articles in current journals as appended to this review.

Dr. Fetterolf was active in many organizations. In the College of Physicians he served at varying times on the Committee of the Mutter Museum, as Chairman both of the Executive Committee and its Section on Otology and Laryngology, as a member of the College Council. He belonged to the American Association of Anatomists, American Laryngological, Rhinological and Otolological Society, and he was Secretary and Treasurer of the John Morgan Society. Perhaps he was most devoted to the American Laryngological Association, a group of men of very high standards in the specialty. He served on the Committee on Tonsil Investigation, first as member and then as Chairman, was First Vice-President in 1922, Treasurer from 1923 to 1932, and had he lived a few more months would have presided as its honored President at the coming annual meeting. In fact, to those of us who realized his loyalty to this body, it almost seemed as though he fought to live long enough to complete this last and greatest service.

His interests aside from medicine were many. We find his College and Medical Fraternity affiliation included Beta Theta Pi (College), Phi Alpha Sigma (Medical), Theta Nu Epsilon (Sophomore), Alpha Omega Alpha (Honorary Medical), Society of Sigma Xi (Honorary Scientific), and Masonic Lodge No. 51. His social organizations had included the Art Club, Merion Cricket, Racquet, Union League and Swiftwater Preserve, all in and about Philadelphia.

To those of us who were Dr. Fetterolf's most intimate friends in his later years, his home life seemed singularly beautiful and happy. His second marriage, on May 26, 1915, to Miss Lila C. Prosser, who survives him, the charming and gifted daughter of Dr. Prosser of Eugene, Oregon, a McGill University man who had been honored by both Dublin and Edinburgh, made his home

one of scientific and intellectual harmony. Both were passionately fond of music, attended opera and the Philadelphia Orchestra and were intimate friends of the famous patron of the latter, Edward Bok. History even records that Dr. Fetterolf organized and led the famous Banjo Club of his college years. His library was a choice one, Kipling's first editions, a complete collection of Bishop's etchings and ancient lore on fishing. Dr. Fetterolf was a real sportsman, loved his golf and was a dry fly enthusiast. The same meticulous care and system were exercised in his play-hobbies as in his medical work. His fishing paraphernalia was as elaborate an equipment and as carefully arranged as his instruments, and his technic as scientifically studied out as his operative procedures.

The impressions given one who has had a long acquaintance and been actively associated with him in recent years are vivid. The earliest impression was that of his care and precision in anatomic work, combined with an exceedingly genial and pleasant nature. College men loved to go to him, confide in him and obtain his advice. In his service to the College of Physicians and the Otolaryngological Chair of the University, all were impressed by the painstaking preparation of his instruction work, his careful organization of the University service, his stimulating influence toward research work among his associates, his keen desire to promote the progress and obtain preferment for his associates. At a recent dinner the Chief Resident of the Hospital characterized Dr. Fetterolf's influence at the University in a very unique expression: "Dr. Fetterolf was a perfect gentleman and most exacting in the requirements of his service." Both of these attributes contributed much in developing the fine corps of assistants left to carry on his work at the University. We must echo the tribute of the Class of 1929 at the University of Pennsylvania Medical School who dedicated their year book, "The Scope," to Dr. Fetterolf.

JAMES A. BABBITT.

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"Frozen Sections from a Case of Sternum-Eroding Aortic Aneurysm." (With George W. Norris, A. B., M. D.) Transactions of the Association of American Physicians, 1919.

"The Otolaryngologic Features of the Influenza Epidemic at Camp Hancock, Georgia, September-December, 1918." (With William J. Rideout, M. D.) ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY for March, 1920.

"Frozen Sections from a Case of Protruding Aneurysm of the Arch of the Aorta." (With George W. Norris, A. B., M. D.) Archives of Internal Medicine, July, 1920, Vol. XXVI, pp. 114-124.

"A Case of Post-Tonsillectomy Pulmonary Abscess." The Laryngoscope, St. Louis, July, 1922.

"The Safeguarding of the Tonsil and Adenoid Operation." American Journal of the Medical Sciences, December, 1922, No. 6, Vol. CLXIV, p. 884.

"A New Instrument for the Insertion of Radium Needles into the Tongue, Pharynx and Larynx." Transactions of the College of Physicians of Philadelphia, Section on Otolaryngology, 1922.

"The Reaction of the Paratonsillar Tissues to Tonsillectomy. A Study in the Etiology of Post-tonsillectomy Pulmonary Abscess." (With Herbert Fox, M. D.) American Journal of the Medical Sciences, December, 1923, No. 6, Vol. CLXVI, p. 802.

"The Use of the Galvano-Cautery in the Treatment of Tuberculosis of the Larynx." The Laryngoscope, St. Louis, April, 1924.

"Ephedrin Sulphate, the Alkaloid of MaHuang." (With Marshall B. Sponsler, M. D.) Archives of Otolaryngology, August, 1925, Vol. 2, pp. 132-135.

"Disease of the Upper Respiratory Tract in Relation to the Etiology and Treatment of Bronchial Asthma." (With Simon S. Leopold, M. D.) Atlantic Medical Journal, February, 1927.

"A Comparison of the Outlines of the Frontal Sinus in Vivo as Shown by Transillumination and the Roentgen Rays." (With Edward E. Sprenkel, M. D.) Archives of Otolaryngology, February, 1929, Vol. 9, pp. 181-184.

"A Case of Infection by *Brucella Melitensis* Var. Abortus Complicating Tonsillectomy." ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY, September, 1929, Vol. XXXVIII, No. 3, p. 675.

Abstracts of Current Articles.

NOSE.

"Experimental Surgery of the Nose and Sinuses." 1. Changes in the Morphology of the Epithelium Following Variations in Ventilation.

Hilding, Anderson (Rochester, Minn.), *Archives of Otolaryngo.*, 16: 9 (July), 1932.

Hilding discusses the different forms of epithelium found in the nose and sinuses. Previous studies have suggested that currents or eddies of air may influence the type of epithelium and this study is done on dogs and rabbits to determine whether such changes in epithelium form can be produced by variations in ventilation.

It was suggested from these experiments that the different forms of epithelium found in the upper part of the respiratory tract are not fixed types. The sheltered cuboidal epithelium of a sinus was seen to change to columnar and stratified with exposure. No similar regression was noted on less exposure to air.

These experiments indicate that the squamous epithelium found in the upper respiratory tract are caused by forceful impact of air at exposed points.

TOBEY.

PHARYNX.

"Ludwig's Angina." Intra-oral Incision in Infections of the Floor of the Mouth.

Houser, K. M. (Philadelphia), *Archives of Otolaryngo.*, 16: 317 (Sept.), 1932.

The definitions all agree that the sublingual space must be involved to constitute true Ludwig's angina. The condition is that of a cellulitis and not a lymphadenitis. In eleven of eighteen cases reported by him a definite history of dental caries was associated. He gives a very clear demonstration of the anatomy showing that it is not strange that Ludwig's angina should act as it does. He strongly advocates the intra-oral operation, an incision being made as early as possible if the diagnosis is confirmed. The site of the incision, longitudinal to the tongue and at about the level of the second molar tooth. The hemostat is then inserted and

passed inward, downward will, in the majority of cases, open up the abscess. In later cases, where there is already an infection in the neck, the external may be combined with the intra-oral operation. His conclusions are based upon fifteen cases with recovery of fourteen.

TOBEY.

Radium Therapy of Inoperable Pharyngolaryngeal Cancer (Le traitement curiethérapique des cancers pharyngolaryngés inopérables).

Bleicher, M. (Nancy), Rev. de Lar. Otol., Rhin., 53: 1176 (Nov.), 1932.

To avoid the annoyance of a loose radium capsule in the hypopharynx, introduced in a rubber catheter via the nostril of the affected side, Bleicher has devised a rubber-covered wire frame which fits the vertical section of the pharynx and nasopharynx, and has the capsule attached to the lower, shortened end of one side of the frame. The frame is laterally compressed by a forceps, its rounded upper end slipped up behind the soft palate to the vomer; then it is allowed to spring apart, and will hold the rubber-covered capsule in close apposition to a neoplasm of the base of the tongue, pyriform sinus or lateral pharyngeal wall, for several days without discomfort in breathing or swallowing.

FENTON.

Paralytic Syndromes in the Cranial and Cervical Sympathetic Nerves in Malignant Neoplasms of the Nasopharynx (Les syndromes paralytiques des nerfs craniens et du sympathique cervical dans les néoplasies malignes du nasopharynx).

Bonnahon, Dr. (Montpellier), Rev. de L. R. O., 53: 595 (May), 1932.

This authoritative study from Prof. Terracol's clinic points out that malignancy of the nasopharynx rarely extends downward, and is implanted (1) about the pharyngeal ostium of the eustachian tube; (2) in the vault of the nasopharynx; (3) along the posterior edge of the vomer and about the choanæ; (4) and the posterior wall of the nasopharynx. They develop usually neither toward the mouth nor the nose, but toward the endocranium or along the base of the skull, following either the lines of closure of the branchial arches, or traveling upward through the posterior ethmoids and sphenoids toward the apices of the petrous bones. Several observations confirm the theory that invasion is by continuity along lymph channels which follow branches of the inferior palatine and inferior pharyngeal arteries

through the anterior lacerated foramen. The peritubal fascia is early involved, with corresponding involvement of deep cervical glands, especially about the exit of the internal jugular.

Syndromes observed include: (1) that of the petrosphenoid crossroads (Jacod), (a) unilateral trigeminal neuralgia, (b) total unilateral ophthalmoplegia, (c) unilateral amaurosis; (2) the Lannois-Gradenigo tumor syndrome, similar to that of petrous tip infection, (a) paralysis or paresis of the abducens, (b) frontoparietal neuralgia of Gasserian origin; (3) Vernet's posterior lacerated foramen syndrome, (a) glossopharyngeal paralysis (upper pharyngeal constrictor), troubles in swallowing, taste disturbances in the posterior third of the tongue, (b) pneumogastric paralysis (sensory) being hypo-, hyper- or paresthesia of the palate, pharynx and larynx with spasmodic cough, (c) spinal accessory paralysis, causing hemipalatal and hemilaryngeal paralysis, and associated paralysis and atrophy of the trapezius and sterno-cleido-mastoid; (4) Collet-Sicard's syndrome of the anterior condyloid and posterior lacerated foramina—which adds to Vernet's symptoms hypoglossal paralysis, with hemiatrophy of the tongue and deviation of its tip toward the affected side; (5) Villaret's syndrome of the retroparotid space—adding pressure paralysis of the cervical sympathetic to the last two syndromes, the widened palpebral fissure, dilated pupil and enophthalmos of Claude Bernard-Horner are thus added symptoms; (6) finally, total unilateral paralysis of all cranial nerves (Froment-Colrat-Dechaume), without papilledema or signs of intracranial pressure, and without sensory or motor disturbances in the limbs.

Careful scrutiny of the vault of the pharynx, especially when unaccountable bleeding or postnasal blocking becomes evident, is recommended.

FENTON.

LARYNX.

"Laryngeal Tuberculosis." Some Experiences and Observations.

Wilkinson, R. W. (*Washington, D. C.*), *Archives of Otolaryngo.*, 16: 331 (Sept.), 1932.

As a result of his experiences and observations at the Kings County Hospital and the Metropolitan Hospital in New York, the author comes to the following conclusions: (1) that the treatment

of laryngeal tuberculosis offers encouraging results in many or most early cases; (2) that it should be brought to the attention of general practitioners that every patient with pulmonary tuberculosis ought to have a laryngeal examination at frequent intervals; (3) the treatment is best carried out in a sanatorium where the pulmonary and laryngeal condition may be under careful surveillance, and (4) that the most effective treatment today is electro-cauterization, vocal rest and sunlight or modified sunlight. We wish to compliment the author on his most complete bibliography on this subject comprising seventy-one references. TOBEY.

Paralysis of the Vocal Cords: A Study of Two Hundred and Seventeen Medical Cases.

New, G. B., and Childrey, J. H. (Rochester, Minn.), *Archives of Otolaryngo.*, :16 (Aug.), 1932.

The authors discuss Semon's law and do not accept it. They have studied 217 cases of paralysis of the vocal cords at the Mayo Clinic. There were three congenital cases; twenty-four due to intracranial lesions; six to toxic neuritis; thirteen cases with jugular foramen syndrome; thirty-four with cancer of the esophagus or hypopharynx; syphilis, ten; cervical tumors, four; goiter, benign, thirty-two; malignant, ten; trauma, four cases; mediastinal disease, twenty-nine; aortic aneurysm, twenty-four; cardiac lesions, ten; tuberculosis, eleven.

The authors found the left cord to be more than twice as often affected. Twenty-six cases were followed for some years and these are reported.

They conclude (1) that paralyzed vocal cords in the cadaveric position usually change to the median line position in a few months, with improvement in the voice and increasing dyspnea, if bilateral; (2) vocal cords in the median line may return to normal function, but most remain there; (3) none changed from median line to cadaveric position. TOBEY.

EAR.

Treatment of Apicitis (Petrositis).

Frencker, P. (Stockholm), *Acta Otolar.*, 17: (1)97, 1932.

Recognizing the technical difficulties in the Kopetzky-Almour approach, Frencker cites two cases in which he followed the route suggested by Holmgren through the concavity of the superior

semicircular canal, after radical operation. He recommends careful mapping of the cell structure by stereoscopic radiographs before operative procedure is planned. Numerous pictures disclose the method of healing by new bone deposition. FENTON.

"Development of the Otic Capsule" 1. Resorption of the Cartilage in the Canal Portion of the Otic Capsule in Human Fetuses and Its Relation to the Growth of the Semicircular Canals.

Bast, T. H. (Madison, Wis.), *Archives of Otolaryngo.*, 16: 19 (July), 1932.

The author reviews other work on the subject and bases his observations on serial sections of ears of fifty-two human fetuses ranging from eight weeks to full term. There are illustrations of microscopic and gross anatomy.

(1) He believes that the growth of the semicircular canals in their cartilage bed involves two processes: (a) Cartilage destruction by necrosis and connective tissue ingrowths; (b) a regressive differentiation of cartilage at advancing margin and a building of cartilage at the receding margin.

(2) Removal of cartilage is accomplished by monocytes or histiocytes.

(3) The canals reach their approximate maximum growth at about half term. TOBEY.

The First Hundred Cases of Mastoiditis Operated Upon by Torrini's Method (I primi cento casi di mastoidite acuta operati col metodo Torrini).

Attore, G. (Florence), *Arch. Ital. di Otol.*, 43: 398 (July), 1932.

Excluding cases which involve the tegmen or expose the middle fossa or the sigmoid sinus, Torrini (1928) recommended immediate suture of the entire wound, leaving filiform drainage below for a day or two. In this series his assistant describes their present technic, by which a small opening for drainage is maintained by a gauze wick into the outer end of the posterior wall of the external auditory meatus. Of these one hundred cases, eighty-one had the sinus exposed; twenty-one had the entire tip removed because of extensive osteomyelitic changes; twenty-eight had the meninges exposed for various reasons; in fourteen cases the lesions had reached the digastric fossa. At the time of the report eighty-eight cases were well and twelve were on the road to recovery. Three cases had to be reopened, after which they got

well; the first was found to present a small area of osteitis in the sinuso-cerebellar angle; the second required removal of a few deep zygomatic cells; the third had a stitch abscess near the upper angle of the incision with deep muscular infiltration. Time of complete healing varied from thirty-two to ninety days; the latter was one of jugular ligation after scarlet fever. The average was fifty-four days.

Advantages claimed include removal of the bandage within ten to fifteen days, with only a bit of cotton in the canal, and the absence of disfiguring scars or depressions behind the ear.

FENTON.

MISCELLANEOUS.

Allergic Headache.

Eyermann, C. H. (*St. Louis*), *J. Allergy*, 2: 106, 1931.

This report is based on a study of sixty-three cases, in which the presenting symptom was headache. In forty-four of these cases the headache was improved when certain and specific foods were omitted from the diet and recurred when these foods were eaten deliberately. In the remaining nineteen cases the headache could not be produced after repeated trials. In thirty-nine of the group of forty-four cases a positive history of allergy was obtained in the antecedents or the children of the patient.

There were allergic manifestations in the patients themselves in forty-three of the forty-four cases. These manifestations were always multiple and the nasal symptoms occurred most frequently. All these patients gave positive skin tests. Borderline positive reactions were considered because clinical experience proved them to be significant in a large percentage of cases. The largest positives were noted in those cases with complicating nasal manifestations and the inhalants, such as pollens, animal emanations and orris root, were the offending allergens.

In most instances the headache was located in the frontal regions, beginning usually over the eyes. Sometimes the localization was occipital and sometimes the pain spread over the head, irrespective of the origin. In no instance was a hemicrania produced. The headaches usually last about twenty-four hours, but may remain for four or five days. Nasal reactions, dizziness,

abdominal distress and nausea and vomiting were the most frequent accompanying symptoms. Edema of the eyelids, face and extremities was frequently noted. Scotomata occurred in only one case of allergic headache and in five cases of nonallergic headache. Headache of allergic origin, according to Eyermann, does not correspond to the accepted definition of migraine. It is suggested that the pathology of allergic headache may be analogous to angioneurotic edema and that the headache may be caused by an edema of the brain.

HANSEL.

Pathology and Symptomatology of Headaches Due to Specific Sensitization.

Rinkel, H. J., and Balyeat, R. M. (Oklahoma City, Okla.), J. A. M. A., 99: 806, 1932.

According to Rinkel and Balyeat, the term "migraine," as commonly used, refers chiefly to paroxysmal attacks of hemicrania (occasionally bilateral headache), associated with sensory and motor disturbances. These sensory and motor symptoms, as well as the crossed hemicrania, gastric and cardiac inhibition and vasomotor changes, indicate disease of the cerebral cortex. Thus they define migraine as a paroxysmal disease characterized by hemicrania (also bilateral headache), and symptomatic evidence of cortical involvement. There are headaches caused by hypersensitivity that cannot be classified as migraine since they do not have cortical features. The authors agree with Eyermann that the term allergic headache should be used instead of the term allergic migraine. Headaches of allergic origin should be designated as such and should be specified as being of certain types, such as migraine, the bandlike headache of hay fever, frontal sinus headache, etc.

The authors' report is based on the study of a group of sixty-five cases which were selected because the headache had been produced by the ingestion of foods and had been relieved by dietary manipulation. The clinical analysis of these cases showed the following significant points: 1. The attacks are longer, though less frequent in females, while males have frequent attacks of shorter duration. 2. Attacks with prodromal symptoms, sensory, motor and vasomotor phenomena, hemicrania and a postmigrainous phase were reported by approximately one-half of these patients.

3. Hyperactivity, either physical or mental, is twice as common in women as in men. 4. The menses are the most frequent predisposing factor in women and physical exhaustion in men. 5. Familial headache occurs in a ratio of 3 : 1 to allergic diseases. 6. One-third of these patients knew that their present complaint was produced by certain foods.

The authors consider hemicrania with premonitory phenomena, aura, signs of exhaustion, etc., as the most important clinical form. Unilateral and bifrontal headaches are often noted in patients with nasal allergy and are usually relieved by nasal treatment.

A family history of headache was obtained in twenty-eight (84.4 per cent) of the women and twenty-three (71.8 per cent) of the men. Heredity not only determines the age of onset but the clinical form.

Allergic headache is classified according to the symptomatology and according to the frequency of recurrence of attacks. Symptomatically the headache is classified in two main groups as the migraine type and the type always without sensory, motor and vasomotor symptoms. The latter group is subdivided into those without nasal and those with nasal pathology. The classification according to the frequency of recurrence of attacks is divided into two main groups, as the periodic type and the nonperiodic type. The consideration of recurrence is important because it gives a clue to factors other than sensitization. The classification on the basis of (1) symptomatology, and (2) recurrence is of value in arranging the treatment. The prognosis seems best in those cases that have the clinical features of migraine and in which recurrences are nonperiodic and occur after each ingestion of the offending food.

HANSEL.

Allergic Migraine: A Review of Sixty Cases.

DeGowin, E. L. (Ann Arbor, Mich.), J. Allergy, 3: 557, 1932.

This report is based on the study of a series of sixty cases of migraine seen during the years 1931 and 1932. They were selected for this series with respect to two criteria, first, a clinical history which would satisfy a good clinician of the diagnosis of migraine, and second, a complete routine study.

Each patient was tested with thirty pollens by the scratch method and with 100 foods by the intradermal method, including

epidermal and miscellaneous substances. The patients in all instances were instructed to eliminate only the foods to which they reacted. When too many foods were implicated the diets of Rowe were used. When the patient became free of symptoms all the reacting foods were reintroduced into the diet singly in order to determine which of the foods actually caused symptoms.

DeGowin defines migraine as characterized by severe, paroxysmal headaches associated with various aura and accompanied by nausea, vomiting, visual disturbances and prostration. The location of the headache was not found to be a constant feature. Pure hemicrania was encountered in only a few instances. The presence of nausea and vomiting with the headache was considered an important factor in establishing the diagnosis of migraine.

In 78 per cent of the cases in which data were available there was partial or complete relief by the elimination of foods to which the patient proved sensitive. A study of familial histories shows the incidence of migraine and other allergic manifestations to be high. DeGowin's statistics are in substantial agreement with those of Balyeat and his coworkers and of Eyer mann. HANSEL.

Unusual Cases of Migraine With Special Reference to Treatment.

Goltman, A. M. (Memphis, Tenn.), J. Allergy, 4: 51, 1932.

Goltman defines migraine as a disease characterized by paroxysmal headache which may be facial, ophthalmic, hemicranial or cranial and by nausea, vomiting and quite often abdominal pain, and preceded as a rule by an aura. He considers it hereditary in origin, interchangeable with other allergic diseases and follows the Mendelian law.

Forty-three cases of true migraine were studied from the allergic and general medical viewpoints. The family history of allergic disease was positive in 70 per cent of the cases. In a patient who had had a decompression operation, bulging and vascular pulsations were noted in this area during attacks of headache. This suggested that in migraine there is a vasodilation or swelling of the brain or meninges, or both. From a practical, clinical point of view these headaches are classified as allergic, endocrine, combined and miscellaneous (due to all other causes). Often in these patients the first sign of an oncoming headache is nasal congestion which may be allergic in nature.

Goltman believes that the successful management of these cases is dependent upon the correct classification as to type. Although foods are the causative agents in the majority of cases it is well to remember that they may play only a minor part in quite a few cases. In some cases inhalants may be of significance. Treatment with specific extracts is recommended in some cases and combined therapy in those in which endocrine factors are present.

In the discussion of this report Vaughan suggested that in those cases in which inhalants appeared to be the cause of headache, the possibility of sinus blockage should be considered rather than migraine.

HANSEL.

Osteomyelitis of the Skull Originating in the Temporal Bone.

Wilensky, A. O. (New York), Archives of Otolaryngo., 16: 160 (Aug.), 1932.

The author has recently discussed general osteomyelitis of the skull, in another paper, published by this magazine and in this article confines himself to osteomyelitis caused by pyogenic bacteria and originating in the temporal bone and resulting from (1) trauma, (2) hematogenous infection, (3) extension cases complicating disease of the otologic apparatus. Three proven cases from the Mt. Sinai Hospital and one private case are reported besides several cases from the literature.

He feels that there is a close clinical similarity between diseases of the petrous pyramid and sphenoid bone.

The complications are intracranial and similar to osteomyelitis of the skull bones in general.

TOBEY.

Specialized Vasculosensory Devices in the Organs of Touch, Taste and Smell (*Dipositifs vasosensoriels spécialisés, etc.*).

Valette, M. H. (Bordeaux), Rev. de L. R. O., 53: 553, (May), 1932.

Proceeding from the accepted theories that proper temperature conditions are necessary for the functioning of the nerve endings of special senses, Dr. Valette has conducted a most accurate investigation of the fine capillary network of venules and of the various venous sinuses in the nasal and lingual mucosa. She has determined the existence of definite specialized networks of this type, adapted to secure the proper temperature reaction, about the tactile hairs, circumvallate papillae and foliated papillae of the

rabbit. These vessels are not merely nutritive, but functionally active. Arterial networks, because of thicker walls and higher pressure, are not functionally practicable and are not found. The olfactory mucosa also has special functional venous networks, which are far less extensive than those of the respiratory zone of the nose.

Such vascularization seems reserved to the highly differentiated nerve terminals of the sense organs which are rather exposed; it does not exist in deeply placed sense organs where temperature conditions are equalized, as in the middle ear. Also, where similar vascular structures are found in regions not containing special sense endings (as in the turbinates) their rôle in the stabilization of temperature is evident and has long been recognized.

FENTON.

Lactic Acid Content of the Blood in Experimental and Pathologic Mouth-breathing (Die Milchsäure im Blute bei experimenteller und pathologischer Mundatmung).

Kreewinsch, P. (Perne), *Acta Oto'ar.*, 17: (1)86, 1932.

Considering animal experiments inconclusive for his purpose, Kreewinsch determined in 30 persons with normal nasal breathing, an average lactic acid index of 19.4 mgm. per cent as against 25.9 mgm. per cent in mouth breathers. The alkali reserve in nose breathers is 56.8 per cent; while in mouth breathers it is 52 per cent, showing venous increase and also pH increase over the normal. With artificial and pathological mouth breathing, an increase of 35 per cent in lactic acid over the normal was observed. This evidences a "tissue-dyspnea" and relative anoxemia with probable remote effects upon the central nervous system and with direct effects upon the rate and depth of respiration.

FENTON.

Leprosy of the Ear, Nose and Throat: Observations on More Than Two Hundred Cases in Hawaii.

Pinkerton, F. J. (Honolulu, T. H.), *Archives of Otolaryngo.*, 16: (Sept.), 1932.

This article is based on the observations of more than two hundred cases of leprosy in Hawaii. He comments that hospitalization with hygienic attention to the patient with an early case offer hope for improvement or perhaps, final cure. He concludes that

leprosy is contracted in early childhood and if it is ever to be brought under control, one must investigate child contacts and bring them under immediate treatment. To quote, "Familial and racial frequency is a fact, and every effort should be made to study these aspects of the disease, minute detail being given to frequent examinations and observations of apparently well persons if they have been known to have had contact with a positive case. The new regimen of the Board for the Control of Leprosy in Hawaii is vitally interested in the early case, because experience and training convince us that this is the way to attack leprosy in this territory." This article should be read in toto by all otolaryngologists. TOBEY.

The Influence of Nasal Respiration on the Secretory and Motor Functions of the Alimentary Canal.

Bondarenko, A. T. (Irkutsk), Acta Otolar., 17: (1)37, 1932.

Questioning some 500 patients with rhinopharyngeal disease, decreased appetite was mentioned by 85 per cent. Fifty-two per cent were constipated, 36 per cent had "heartburn," 6 per cent gastric pain, 5 per cent belching, out of a group of 116 who made the most serious complaints. Experiments on isolated loops of intestine in dogs showed slowed peristalsis and slowed breathing; there is also an increase in the caliber of the bowel and decrease in the alkalinity of its contents. FENTON.

Use of Negative Pressure in Otolaryngology: Report of a Serious Accident.

Gordon, W. (Philadelphia), Archives of Otolaryngo., 16: 370 (Sept.), 1932.

The author has correlated the opinions and experiences of a large number of authors and comments that there is a very definite place for suction or negative pressure in the treatment of diseases in otolaryngology, and wisely adds that it should be applied with the utmost care, gentleness and skill. TOBEY.

Radiographic Diagnosis of Gradenigo's Syndrome (Die roentgenographische Diagnose des Syndroms von Gradenigo).

Burger, H. (Amsterdam), Acta Otolar., 17: (4)353, 1932.

This is a careful study of all previously reported roentgenograms of such cases, with pictures of two new ones, best seen in the oblique cerebellar-fossa projection of Stenvers. FENTON.

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